

L5 ANSWER 25 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:759916 CAPLUS
 DN 134:36796
 TI .gamma.-Tocopherol and its major metabolite, in contrast to
 .alpha.-tocopherol, inhibit cyclooxygenase activity in macrophages and
 epithelial cells
 AU Jiang, Qing; Elson-Schwab, Ilan; Courtemanche, Chantal; Ames, Bruce N.
 CS Division of Biochemistry and Molecular Biology, University of California,
 Berkeley, CA, 94720, USA
 SO Proceedings of the National Academy of Sciences of the United States of
 America (2000), 97(21), 11494-11499
 CODEN: PNASA6; ISSN: 0027-8424
 PB National Academy of Sciences
 DT Journal
 LA English
 CC 1-7 (Pharmacology)
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 and vascular heart disease. Here we report that .gamma.-tocopherol
 (.gamma.T) reduced PGE2 synthesis in both lipopolysaccharide
 (LPS)-stimulated RAW264.7 macrophages and IL-1.beta.-treated A549 human
 epithelial cells with an apparent IC50 of 7.5 and 4 .mu.M, resp. The
 major metabolite of dietary .gamma.T, 2,7,8-trimethyl-2-((beta.-
 carboxyethyl)-6-hydroxychroman (.gamma.-CEHC), also exhibited an
 inhibitory effect, with an IC50 of .apprx.30 .mu.M in these cells. In
 contrast, .alpha.-tocopherol at 50 .mu.M slightly reduced (25%) PGE2
 formation in macrophages, but had no effect in epithelial cells. The
 inhibitory effects of .gamma.T and .gamma.-CEHC stemmed from
 their inhibition of COX-2 activity, rather than affecting protein
 expression or substrate availability, and appeared to be independent of
 antioxidant activity. .gamma.-CEHC also inhibited PGE2
 synthesis when exposed for 1 h to COX-2-preinduced cells followed by the
 addn. of arachidonic acid (AA), whereas under similar conditions, .gamma.T
 required an 8- to 24-h incubation period to cause the inhibition. The
 inhibitory potency of .gamma.T and .gamma.-CEHC was diminished
 by an increase in AA concn., suggesting that they might compete with AA at
 the active site of COX-2. We also obsd. a moderate redn. of nitrite
 accumulation and suppression of inducible nitric oxide synthase expression
 by .gamma.T in lipopolysaccharide-treated macrophages. These findings
 indicate that .gamma.T and its major metabolite possess anti-inflammatory
 activity and that .gamma.T at physiol. concns. may be important in human
 disease prevention.
 ST gamma tocopherol cyclooxygenase 2 antiinflammatory
 IT Antioxidants
 (pharmaceutical; .gamma.-Tocopherol and its major metabolite inhibit
 cyclooxygenase activity in macrophages and epithelial cells)
 IT 39391-18-9
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (cyclooxygenase-2; .gamma.-Tocopherol and its major metabolite inhibit
 cyclooxygenase activity in macrophages and epithelial cells)
 IT 59-02-9, .alpha.-Tocopherol
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological
 study, unclassified); BIOL (Biological study)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase
 activity in macrophages and epithelial cells)
 IT 178167-88-9
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological
 study, unclassified); MFM (Metabolic formation); THU (Therapeutic use);
 BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase
 activity in macrophages and epithelial cells)

IT 7616-22-0, .gamma.-Tocopherol
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 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 363-24-6, PGE2 41598-07-6, PGD2 125978-95-2, Nitric oxide synthase
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 155976-51-5, 8-Isoprostane
 RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
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AN 2000:788255 CAPLUS
 DN 134:55952
 TI Production of LLU-.alpha. following an oral administration of
 .gamma.-tocotrienol or .gamma.-tocopherol to rats
 AU Hattori, Akihiro; Fukushima, Takeshi; Yoshimura, Hiroyuki; Abe, Kouichi;
 Imai, Kazuhiro
 CS Department of Bio-Analytical Chemistry, Graduate School of Pharmaceutical
 Sciences, The University of Tokyo, Tokyo, 113-0033, Japan
 SO Biological & Pharmaceutical Bulletin (2000), 23(11), 1395-1397
 CODEN: BPBLEO; ISSN: 0918-6158
 PB Pharmaceutical Society of Japan
 DT Journal
 LA English
 CC 18-2 (Animal Nutrition)
 Section cross-reference(s): 13
 AB An oral administration of .gamma.-tocotrienol (.gamma.-T3) or
 .gamma.-tocopherol (.gamma.-Toc) to male rats caused an increase of the
 concn. of 2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxy chroman
 (LLU-.alpha., .gamma.-CEHC), a natriuretic compd., in plasma
 with a Tmax of 9 h. The configuration at C-2 of LLU-.alpha. produced from
 .gamma.-T3 or .gamma.-Toc was assigned as S-form by an HPLC equipped with
 a chiral column. These data indicated that LLU-.alpha. was produced not
 only from .gamma.-Toc but also .gamma.-T3, without racemization at C-2 in
 rats.
 ST LLU tocotrienol tocopherol metab; chroman deriv formation tocotrienol
 tocopherol; hydroxychroman deriv formation tocotrienol tocopherol
 IT 7616-22-0, .gamma.-Tocopherol 14101-61-2, .gamma.-Tocotrienol
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (LLU-.alpha. prodn. following an oral administration of
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 IT 178167-88-9
 RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL
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 IT 7616-22-0, .gamma.-Tocopherol

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IT 363-24-6, PGE2 41598-07-6, PGD2 125978-95-2, Nitric oxide synthase
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RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

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AN 1999:504149 CAPLUS
 DN 131:252216
 TI Chemopreventive activity of celecoxib, a specific cyclooxygenase-2 inhibitor, and indomethacin against ultraviolet light-induced **skin** carcinogenesis
 AU Fischer, Susan M.; Lo, Herng-Hsang; Gordon, Gary B.; Seibert, Karen; Kelloff, Gary; Lubet, Ronald A.; Conti, Claudio J.
 CS Science Park-Research Division, The University of Texas M. D. Anderson Cancer Center, Smithville, TX, 78957, USA
 SO Molecular Carcinogenesis (1999), 25(4), 231-240
 CODEN: MOCAE8; ISSN: 0899-1987
 PB Wiley-Liss, Inc.
 DT Journal
 LA English
 CC 1-6 (Pharmacology)
 Section cross-reference(s): 8
 AB Epidemiol. and dietary studies suggest that nonsteroidal anti-inflammatory drugs (NSAIDs) reduce the risk of colon cancer, possibly through a mechanism involving inhibition of cyclooxygenase (COX)-2, which is overexpressed in premalignant adenomatous polyps and colon cancer. Because UV light (UV) can induce COX-2 and nonspecific NSAIDs can decrease UV-induced **skin** cancer, we evaluated the ability of two compds., celecoxib (a specific COX-2 inhibitor) and indomethacin (a nonspecific NSAID), to block UV-induced **skin** tumor development in SKH:HR-1-hrBr hairless mice. Mice fed 150 or 500 ppm celecoxib showed a dose-dependent redn. (60% and 89%, resp.) in tumor yield. Indomethacin (4 ppm) reduced tumor yield by 78%. Although both acute and chronic UV exposure increased cell proliferation and edema, neither compd. reduced these parameters. In contrast, UV-induced prostaglandin synthesis in the epidermis was effectively blocked by both compds. UV-induced increases in COX-2 expression in **skin** were also not altered in any of the treatment groups. Similarly, tumors that constitutively express high levels of COX-2 displayed no redn. by treatment with celecoxib or indomethacin. The dramatic protective effects of celecoxib suggests that specific COX-2 inhibitors may offer a way to safely reduce the risk of **skin** cancer in humans.
 ST COX2 celecoxib NSAID indomethacin UV **skin** carcinogenesis
 IT Radioprotectants
 Transformation, neoplastic
 UV radiation
 (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced **skin** carcinogenesis)
 IT Prostaglandins
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced **skin** carcinogenesis: epidermal prostaglandin synthesis inhibition)
 IT **Skin**
 (epidermis; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced **skin** carcinogenesis: epidermal prostaglandin synthesis inhibition)
 IT **Skin, neoplasm**
Skin, neoplasm
 (inhibitors; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced **skin** carcinogenesis)
 IT Anti-inflammatory agents
 (nonsteroidal; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced **skin** carcinogenesis)

IT Antitumor agents
Antitumor agents
(**skin**; **COX-2** inhibitor celecoxib and
NSAID indomethacin prevention of UV light-induced **skin**
carcinogenesis)

IT 39391-18-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(2; **COX-2** inhibitor celecoxib and NSAID
indomethacin prevention of UV light-induced **skin**
carcinogenesis)

IT 53-86-1, Indomethacin 169590-42-5, Celecoxib
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES
(Uses)
(**COX-2** inhibitor celecoxib and NSAID indomethacin
prevention of UV light-induced **skin** carcinogenesis)

IT 363-24-6, Pge2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(**COX-2** inhibitor celecoxib and NSAID indomethacin
prevention of UV light-induced **skin** carcinogenesis: epidermal
prostaglandin synthesis inhibition)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
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1991, VII, P1365

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L21 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2003 ACS
 AN 2001:870069 CAPLUS
 DN 136:130852
 TI Rays and arrays: the transcriptional program in the response of human epidermal keratinocytes to UVB illumination
 AU Li, Deling; Turit, Thomas G.; Schuck, Alyssa; Freedberg, Irwin M.; Khitrov, Gregory; Blumenberg, Miroslav
 CS The R. O. Perelman Department of Dermatology, New York University School of Medicine, New York, NY, USA
 SO FASEB Journal (2001), 15(13), 2533-2535, 10.1096/fj.01-0172fje
 CODEN: FAJOEC; ISSN: 0892-6638
 PB Federation of American Societies for Experimental Biology
 DT Journal
 LA English
 CC 8-7 (Radiation Biochemistry)
 AB The epidermis, our first line of defense from UV light, bears the majority of photodamage, which results in skin thinning, wrinkling, keratosis, and malignancy. Hypothesizing that skin has specific mechanisms to protect itself and the organism from UV damage, we used DNA arrays to follow UV-caused gene expression changes in epidermal keratinocytes. Of the 6,800 genes examd., UV regulates the expression of at least 198. Three waves of changes in gene expression can be distinguished, 0.5-2, 4-8, and 16-24 h after illumination. The first contains transcription factors, signal transducing, and cytoskeletal proteins that change cell phenotype from a normal, fast-growing cell to an activated, paused cell. The second contains secreted growth factors, cytokines, and chemokines; keratinocytes, having changed their own physiol., alert the surrounding tissues to the UV damage. The third wave contains components of the cornified envelope, as keratinocytes enhance the epidermal protective covering and, simultaneously, terminally differentiate and die, removing a carcinogenic threat. UV also induces the expression of mitochondrial proteins that provide addnl. energy, and the enzymes that synthesize raw materials for DNA repair. Using a novel skin organ culture model, we demonstrated that the UV-induced changes detected in keratinocyte cultures also occur in human epidermis in vivo.
 ST UVB radiation regulated gene skin keratinocyte
 IT Macrophage inflammatory protein 2
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (MIP-2.alpha.; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)
 IT Cell membrane
 Cytoskeleton
 DNA repair
 Post-transcriptional processing
 Signal transduction, biological
 (UVB-regulated genes in human epidermal keratinocytes)
 IT Chemokines
 Cytokines
 Elastins
 Gene, animal
 Growth factors, animal
 Interleukin 8
 Transcription factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (UVB-regulated genes in human epidermal keratinocytes)
 IT Transcription factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (c-myc; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)
 IT Cell envelope
 (cornified; UVB-regulated genes in human epidermal keratinocytes)
 IT Antioxidants

(defense proteins; UVB-regulated genes in human epidermal keratinocytes)

IT Cell junction
(desmosome; UVB-regulated genes in human epidermal keratinocytes)

IT Metabolism
(energy; UVB-regulated genes in human epidermal keratinocytes)

IT Skin
(epidermis; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(genes regulated by; UVB-regulated genes in human epidermal keratinocytes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(involucrins; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Skin
(keratinocyte; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Chemokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(melanoma growth-stimulating activity-.beta.; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Skin, disease
(photoaging; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Post-translational processing
(proteolytic; UVB-regulated genes in human epidermal keratinocytes)

IT DNA microarray technology
Human
UV B radiation
(transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT mRNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Caseins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.beta.-; UVB-regulated genes in human epidermal keratinocytes)

IT 329900-75-6, **Cyclooxygenase 2**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

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AN 2001:800060 CAPLUS
 DN 136:66317
 TI UV erythema reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined in comparison to indomethacin
 AU Grundmann, Jens-Uwe; Bockelmann, Raik; Bonnekoh, Bernd; Gollnick, Harald P. M.
 CS Department of Dermatology and Venereology, Otto-von-Guericke-University, Magdeburg, D-39120, Germany
 SO Photochemistry and Photobiology (2001), 74(4), 587-592
 CODEN: PHCBAP; ISSN: 0031-8655
 PB American Society for Photobiology
 DT Journal
 LA English
 CC 8-9 (Radiation Biochemistry)
 Section cross-reference(s): 1
 AB UV light exerts hazardous effects such as induction of skin cancer and premature **skin aging**. In this study we evaluated an assumptive anti-inflammatory effect of the nonsedative histamine H1-receptor antagonist, mizolastine, on UV-induced acute sunburn reaction. Therefore, a clin., randomized, double-blind, four-arm, crossover study was conducted in healthy young female volunteers (skin type II) comparing the UV sensitivity under mizolastine, acetyl-salicylic acid (ASA), indomethacin or a mizolastine/ASA combination. Moreover, HaCaT keratinocytes were incubated with mizolastine under various UV treatment modalities in vitro to study its effect on the release of inflammatory cytokines, i.e. interleukin (IL)-1.alpha., IL-6 and tumor necrosis factor .alpha. (TNF-.alpha.). All three drugs were effective in suppressing the UVB-, UVA- and combined UVA/UVB-erythema. However, the strongest effects were obsd. using the combined treatment with both 250 mg ASA and 10 mg mizolastine. An inhibitory effect in vitro of 10 nM mizolastine upon UV-induced cytokine release from HaCaT keratinocytes was obsd. for IL-1.alpha. at 24 h after 10 J/cm2 UVA1, for IL-6 at 48 h after 10 J/cm2 UVA1 and 30 mJ/cm2 UVB, and also for TNF-.alpha. at 4 h after 10 J/cm2 UVA, 10 J/cm2 UVA1 and 30 mJ/cm2 UVB, resp. The combination of mizolastine and ASA can be strongly recommended as a protective measure against UV erythema development with a lower unwanted side effect profile than that of the hitherto treatment modality, i.e. indomethacin.
 ST UV erythema protection antiinflammatory mizolastine acetylsalicylic acid cytokine
 IT Anti-inflammatory agents
 Drug interactions
 Erythema
 Human
 Radioprotectants
 Sunburn
 UV A radiation
 UV radiation
 (UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)
 IT Interleukin 1.alpha.
 Interleukin 6
 Tumor necrosis factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)
 IT Skin, disease
 (aging, prevention; UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)
 IT Cytokines
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (inflammatory; UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)

IT Skin
 (keratinocyte; UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)

IT 50-78-2, Acetyl-salicylic acid 108612-45-9, Mizolastine
 RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)

IT 39391-18-9, **Cyclooxygenase**
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (inhibitors; UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
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- (38) Toth-Jakatics, R; Hum Pathol 2000, V31, P955 MEDLINE
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- (40) Vargaftig, B; Int Acad Biomed Drug Res 1993, V6, P27
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NEWS	30	Apr 11	Display formats in DGENE enhanced
NEWS	31	Apr 14	MEDLINE Reload
NEWS	32	Apr 17	Polymer searching in REGISTRY enhanced
NEWS	33	Jun 13	Indexing from 1947 to 1956 added to records in CA/CAPLUS
NEWS	34	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX
NEWS	35	Apr 28	RDISCLOSURE now available on STN
NEWS	36	May 05	Pharmacokinetic information and systematic chemical names added to PHAR
NEWS	37	May 15	MEDLINE file segment of TOXCENTER reloaded
NEWS	38	May 15	Supporter information for ENCOMPPAT and ENCOMPLIT updated
NEWS	39	May 16	CHEMREACT will be removed from STN
NEWS	40	May 19	Simultaneous left and right truncation added to WSCA
NEWS	41	May 19	RAPRA enhanced with new search field, simultaneous left and right truncation
NEWS	42	Jun 06	Simultaneous left and right truncation added to CBNB
NEWS	43	Jun 06	PASCAL enhanced with additional data

NEWS 44 Jun 20 2003 edition of the FSTA Thesaurus is now available
NEWS 45 Jun 25 HSDB has been reloaded

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MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
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Experimental and calculated property data are now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
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=> s chec

L1 15 CHEC

=> d l1 15

L1 ANSWER 15 OF 15 REGISTRY COPYRIGHT 2003 ACS

RN 156288-21-0 REGISTRY

CN Protein (Bacillus subtilis 209-amino acid) (9CI) (CA INDEX NAME)

OTHER NAMES:

CN **Protein (Bacillus subtilis gene cheC)**
CN Protein orfA (Bacillus subtilis fla/che region)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS

****RELATED SEQUENCES AVAILABLE WITH SEQLINK****

***** STRUCTURE DIAGRAM IS NOT AVAILABLE *****
***** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE *****
2 REFERENCES IN FILE CA (1957 TO DATE)
2 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=> s 11 1
MISSING OPERATOR

=> d 11 1

L1 ANSWER 1 OF 15 REGISTRY COPYRIGHT 2003 ACS
RN 500491-62-3 REGISTRY
CN **Chemotaxis protein cheC (Clostridium tetani strain E88 gene cheC)**
(9CI) (CA INDEX NAME)

OTHER NAMES:

CN GenBank AAO36263
CN GenBank AAO36263 (Translated from: GenBank AE015942)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: CA, CAPLUS, TOXCENTER

***** STRUCTURE DIAGRAM IS NOT AVAILABLE *****
***** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE *****
1 REFERENCES IN FILE CA (1957 TO DATE)
1 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=> s cehc
L2 5 CEHC

=> d 12 5

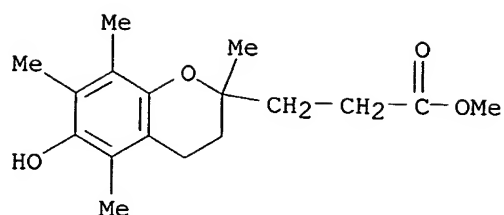
L2 ANSWER 5 OF 5 REGISTRY COPYRIGHT 2003 ACS
RN 98760-50-0 REGISTRY
CN 2H-1-Benzopyran-2-propanoic acid, 3,4-dihydro-6-hydroxy-2,5,7,8-tetramethyl-, methyl ester (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 2-Chromanpropionic acid, 6-hydroxy-2,5,7,8-tetramethyl-, methyl ester (6CI)

OTHER NAMES:

CN **.alpha.-CEHC methyl ester**
FS 3D CONCORD
MF C17 H24 O4
SR CA
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2 REFERENCES IN FILE CA (1957 TO DATE)
 2 REFERENCES IN FILE CAPLUS (1957 TO DATE)
 2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file caplus
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ENTRY	SESSION
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=> s l2
 L3 2 L2

=> d his

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L1 15 S CHEC
 L2 5 S CEHC

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L3 2 S L2

=> s l2
 L4 2 L2

=> d 14 1-2

L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
AN 2002:668559 CAPLUS
DN 138:3996
TI Synthesis and analysis of conjugates of the major vitamin E metabolite, .alpha.-CEHC
AU Pope, Simon A. S.; Burtin, Guillaume E.; Clayton, Peter T.; Madge, David J.; Muller, David P. R.
CS The Institute of Child Health, Biochemistry, Endocrinology and Metabolism Unit, University College London, London, UK
SO Free Radical Biology & Medicine (2002), 33(6), 807-817
CODEN: FRBMEH; ISSN: 0891-5849
PB Elsevier Science Inc.
DT Journal
LA English
OS CASREACT 138:3996
RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
AN 1985:615586 CAPLUS
DN 103:215586
TI Autoxidation of biological molecules. 4. Maximizing the antioxidant activity of phenols
AU Burton, G. W.; Doba, T.; Gabe, E.; Hughes, L.; Lee, F. L.; Prasad, L.; Ingold, Keith U.
CS Div. Chem., Natl. Res. Counc. Canada, Ottawa, ON, K1A 0R6, Can.
SO Journal of the American Chemical Society (1985), 107(24), 7053-65
CODEN: JACSAT; ISSN: 0002-7863
DT Journal
LA English
OS CASREACT 103:215586

=> s cehc

L5 41 CEHC

=> d 15 20-41

L5 ANSWER 20 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2001:465748 CAPLUS
DN 135:194909
TI .alpha.-tocopherol affects the urinary and biliary excretion of 2,7,8-trimethyl-2(2'-carboxyethyl)-6-hydroxychroman, .gamma.-tocopherol metabolite, in rats
AU Kiyose, Chikako; Saito, Hisako; Kaneko, Kazuyo; Hamamura, Kimio; Tomioka, Mitsugu; Ueda, Tadahiko; Igarashi, Osamu
CS Institute of Environmental Science for Human Life, Ochanomizu University, Tokyo, 112-8610, Japan
SO Lipids (2001), 36(5), 467-472
CODEN: LPDSAP; ISSN: 0024-4201
PB AOCs Press
DT Journal
LA English
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 21 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2001:251669 CAPLUS
DN 134:290494

TI A fluorimetric, column-switching HPLC and its application to an
elimination study of LLU-.alpha. enantiomers in rat plasma
AU Hattori, Akihiro; Fukushima, Takeshi; Hamamura, Kimio; Kato, Masaru; Imai,
Kazuhiro
CS Department of Bio-Analytical Chemistry, Graduate School of Pharmaceutical
Sciences, The University of Tokyo, Tokyo, 113-0033, Japan
SO Biomedical Chromatography (2001), 15(2), 95-99
CODEN: BICHE2; ISSN: 0269-3879
PB John Wiley & Sons Ltd.
DT Journal
LA English
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 22 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2001:120242 CAPLUS
DN 134:236753
TI .alpha.- and .gamma.-tocotrienols are metabolized to carboxyethyl-
hydroxychroman derivatives and excreted in human urine
AU Lodge, John K.; Ridlington, James; Leonard, Scott; Vaule, Heather; Traber,
Maret G.
CS Linus Pauling Institute, Oregon State University, Corvallis, OR,
97331-6512, USA
SO Lipids (2001), 36(1), 43-48
CODEN: LPDSAP; ISSN: 0024-4201
PB AOCs Press
DT Journal
LA English
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 23 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2000:895441 CAPLUS
DN 134:192659
TI Bioavailability and potency of natural-source and all-racemic
.alpha.-tocopherol in the human: A dispute
AU Hoppe, P. P.; Krennrich, G.
CS Nutrition Research Station, BASF Aktiengesellschaft, Offenbach, 76877,
Germany
SO European Journal of Nutrition (2000), 39(5), 183-193
CODEN: EJNUFZ; ISSN: 1436-6207
PB Steinkopff Verlag
DT Journal
LA English
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 24 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2000:788255 CAPLUS
DN 134:55952
TI Production of LLU-.alpha. following an oral administration of
.gamma.-tocotrienol or .gamma.-tocopherol to rats
AU Hattori, Akihiro; Fukushima, Takeshi; Yoshimura, Hiroyuki; Abe, Kouichi;
Imai, Kazuhiro
CS Department of Bio-Analytical Chemistry, Graduate School of Pharmaceutical
Sciences, The University of Tokyo, Tokyo, 113-0033, Japan
SO Biological & Pharmaceutical Bulletin (2000), 23(11), 1395-1397
CODEN: BPBLEO; ISSN: 0918-6158
PB Pharmaceutical Society of Japan
DT Journal
LA English
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 25 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:759916 CAPLUS
 DN 134:36796
 TI .gamma.-Tocopherol and its major metabolite, in contrast to
 .alpha.-tocopherol, inhibit cyclooxygenase activity in macrophages and
 epithelial cells
 AU Jiang, Qing; Elson-Schwab, Ilan; Courtemanche, Chantal; Ames, Bruce N.
 CS Division of Biochemistry and Molecular Biology, University of California,
 Berkeley, CA, 94720, USA
 SO Proceedings of the National Academy of Sciences of the United States of
 America (2000), 97(21), 11494-11499
 CODEN: PNASA6; ISSN: 0027-8424
 PB National Academy of Sciences
 DT Journal
 LA English
 RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 26 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:728570 CAPLUS
 DN 134:16205
 TI Urinary .alpha.-tocopherol metabolites in .alpha.-tocopherol transfer
 protein-deficient patients
 AU Schuelke, Markus; Elsner, Angelika; Finckh, Barbara; Kohlschutter,
 Alfried; Hubner, Christoph; Brigelius-Flohe, Regina
 CS Department of Neuropediatrics, Charite University Hospital, Humboldt
 University Berlin, Berlin, D-13353, Germany
 SO Journal of Lipid Research (2000), 41(10), 1543-1551
 CODEN: JLPRAW; ISSN: 0022-2275
 PB Lipid Research, Inc.
 DT Journal
 LA English
 RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 27 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:605350 CAPLUS
 DN 134:2274
 TI A New Method for the Analysis of Urinary Vitamin E Metabolites and the
 Tentative Identification of a Novel Group of Compounds
 AU Pope, S. A. S.; Clayton, P. T.; Muller, D. P. R.
 CS Biochemistry, Endocrinology and Metabolism Unit, Institute of Child
 Health, University College London, London, UK
 SO Archives of Biochemistry and Biophysics (2000), 381(1), 8-15
 CODEN: ABBIA4; ISSN: 0003-9861
 PB Academic Press
 DT Journal
 LA English
 RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 28 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:456026 CAPLUS
 DN 133:149828
 TI Overview of studies on vitamin E metabolism - Missing link of vitamin E
 metabolism
 AU Nakamura, Tetsuya
 CS Department of Chemistry, Shibaura Institute of Technology, Fukasaku,
 Ohmiya, 330-8570, Japan
 SO Bitamin (2000), 74(5-6), 255-261

CODEN: BTMNA7; ISSN: 0006-386X

PB Nippon Bitamin Gakkai
DT Journal; General Review
LA Japanese

L5 ANSWER 29 OF 41 CAPLUS COPYRIGHT 2003 ACS

AN 2000:420456 CAPLUS

DN 133:99955

TI Occurrence and determination of a natriuretic hormone,
2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxy chroman, in rat plasma,
urine, and bile

AU Hattori, Akihiro; Fukushima, Takeshi; Imai, Kazuhiro

CS Department of Bio-Analytical Chemistry, Graduate School of Pharmaceutical
Sciences, University of Tokyo, Tokyo, 113-0033, Japan

SO Analytical Biochemistry (2000), 281(2), 209-215

CODEN: ANBCA2; ISSN: 0003-2697

PB Academic Press

DT Journal

LA English

RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 30 OF 41 CAPLUS COPYRIGHT 2003 ACS

AN 2000:337048 CAPLUS

DN 133:104332

TI Studies of the metabolism of .alpha.-tocopherol stereoisomers in rats
using [5-methyl-14C]SRR- and RRR-.alpha.-tocopherol

AU Kaneko, Kazuyo; Kiyose, Chikako; Ueda, Tadahiko; Ichikawa, Hisatsugu;
Igarashi, Osamu

CS Institute of Environmental Science for Human Life, Ochanomizu University,
Tokyo, 112-8610, Japan

SO Journal of Lipid Research (2000), 41(3), 357-367

CODEN: JLPRAW; ISSN: 0022-2275

PB Lipid Research, Inc.

DT Journal

LA English

RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 31 OF 41 CAPLUS COPYRIGHT 2003 ACS

AN 2000:156210 CAPLUS

DN 132:347067

TI A novel 5'-carboxychroman metabolite of .gamma.-tocopherol secreted by
HepG2 cells and excreted in human urine

AU Parker, Robert S.; Swanson, Joy E.

CS Division of Nutritional Sciences, Cornell University, Ithaca, NY, 14853,
USA

SO Biochemical and Biophysical Research Communications (2000), 269(2),
580-583

CODEN: BBRC9; ISSN: 0006-291X

PB Academic Press

DT Journal

LA English

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 32 OF 41 CAPLUS COPYRIGHT 2003 ACS

AN 2000:51722 CAPLUS

DN 132:248204

TI A rapid method for the extraction and determination of vitamin E
metabolites in human urine

AU Lodge, John K.; Traber, Maret G.; Elsner, Angelika; Brigelius-Flohe,

Regina
CS Linus Pauling Institute, Oregon State University, Corvallis, OR, 97330,
USA
SO Journal of Lipid Research (2000), 41(1), 148-154
CODEN: JLPRAW; ISSN: 0022-2275
PB Lipid Research, Inc.
DT Journal
LA English

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 33 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1999:711893 CAPLUS
DN 132:75508
TI Quantification of the .alpha.- and .gamma.-Tocopherol Metabolites
2,5,7,8-Tetramethyl-2-(2'-carboxyethyl)-6-hydroxychroman and
2,7,8-Trimethyl-2-(2'-carboxyethyl)-6-hydroxychroman in Human Serum
AU Stahl, W.; Graf, P.; Brigelius-Flohe, R.; Wechter, W.; Sies, H.
CS Institut fur Physiologische Chemie I, Heinrich-Heine-Universitat
Dusseldorf, Germany
SO Analytical Biochemistry (1999), 275(2), 254-259
CODEN: ANBCA2; ISSN: 0003-2697
PB Academic Press
DT Journal
LA English

RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 34 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1999:464704 CAPLUS
DN 131:223815
TI Nitric oxide can function as either a killer molecule or an antiapoptotic
effector in cardiomyocytes
AU Stefanelli, Claudio; Pignatti, Carla; Tantini, Benedetta; Stanic, Ivana;
Bonavita, Francesca; Muscari, Claudio; Guarnieri, Carlo; Clo, Carlo;
Caldarera, Claudio M.
CS Department of Biochemistry 'G. Moruzzi', University of Bologna, Bologna,
40126, Italy
SO Biochimica et Biophysica Acta (1999), 1450(3), 406-413
CODEN: BBACAQ; ISSN: 0006-3002
PB Elsevier Science B.V.
DT Journal
LA English

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 35 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1999:437875 CAPLUS
DN 131:198876
TI Vitamin E: function and metabolism
AU Brigelius-Flohe, Regina; Traber, Maret G.
CS German Institute of Human Nutrition, Bergholz-Rehbrücke, D-14558, Germany
SO FASEB Journal (1999), 13(10), 1145-1155
CODEN: FAJOEC; ISSN: 0892-6638
PB Federation of American Societies for Experimental Biology
DT Journal; General Review
LA English

RE.CNT 112 THERE ARE 112 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 36 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1999:235185 CAPLUS

DN 131:31364
TI Urinary excretion of 2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxychroman is a major route of elimination of .gamma.-tocopherol in humans
AU Swanson, J. E.; Ben, R. N.; Burton, G. W.; Parker, R. S.
CS Division of Nutritional Sciences, Cornell University, Ithaca, NY, 14853, USA
SO Journal of Lipid Research (1999), 40(4), 665-671
CODEN: JLPRAW; ISSN: 0022-2275
PB Lipid Research, Inc.
DT Journal
LA English
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 37 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1998:677442 CAPLUS
DN 130:50169
TI Synthetic as compared with natural vitamin E is preferentially excreted as .alpha.-CEHC in human urine: studies using deuterated .alpha.-tocopheryl acetates
AU Traber, Maret G.; Elsner, Angelika; Brigelius-Flohe, Regina
CS Linus Pauling Institute, Oregon State University, Corvallis, OR, 97330, USA
SO FEBS Letters (1998), 437(1,2), 145-148
CODEN: FEBLAL; ISSN: 0014-5793
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 38 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1997:712440 CAPLUS
DN 128:11596
TI .alpha.-Carboxyethyl-6-hydroxychroman as urinary metabolite of vitamin E
AU Schultz, Manfred; Leist, Marcel; Elsner, Angelika; Brigelius-Flohe, Regina
CS USA
SO Methods in Enzymology (1997), 282(Vitamins and Coenzymes, Part L), 297-310
CODEN: MENZAU; ISSN: 0076-6879
PB Academic
DT Journal
LA English

L5 ANSWER 39 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1996:69202 CAPLUS
DN 124:144421
TI Novel urinary metabolite of .alpha.-tocopherol, 2,5,7,8-tetramethyl-2(2'-carboxyethyl)-6-hydroxychroman, as an indicator of an adequate vitamin E supply?
AU Schultz, Manfred; Leist, Marcel; Petrzika, Marion; Gassmann, Berthold; Brigelius-Flohe, Regina
CS Department Vitamins and Atherosclerosis, German Institute Human Nutrition, Potsdam-Rehbrücke, D-14558, Germany
SO American Journal of Clinical Nutrition (1995), 62(6, Suppl.), 1527S-34S
CODEN: AJCNAC; ISSN: 0002-9165
PB American Society for Clinical Nutrition
DT Journal
LA English

L5 ANSWER 40 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1991:427028 CAPLUS

DN 115:27028
TI Serum .gamma.-glutamyl transpeptidase in chronic extrahepatic cholestasis
AU Castro-E-Silva, O., Jr.; Franco, C. F. F.; Souza, M. E. J.; Picinato, M.
A. N. C.; Santos, J. S.; Ceneviva, R.
CS Fac. Med. Ribeirao Preto, Univ. Sao Paulo, Ribeirao Preto, 14049, Brazil
SO Brazilian Journal of Medical and Biological Research (1990), 23(6-7),
515-18
CODEN: BJMRDK; ISSN: 0100-879X
DT Journal
LA English

L5 ANSWER 41 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 1985:497831 CAPLUS
DN 103:97831
TI Preparation and CD spectra of cobalt(III) complexes with
S-(carboxymethyl)-L-cysteinate and its analogs
AU Okamoto, Kenichi; Suzuki, Masutaro; Einaga, Hisahiko; Hidaka, Jinsai
CS Dep. Chem., Univ. Tsukuba, Ibaraki, 305, Japan
SO Bulletin of the Chemical Society of Japan (1985), 58(6), 1807-11
CODEN: BCSJA8; ISSN: 0009-2673
DT Journal
LA English

=> d 15 22 all

L5 ANSWER 22 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2001:120242 CAPLUS
DN 134:236753
TI .alpha.- and .gamma.-tocotrienols are metabolized to carboxyethyl-
hydroxychroman derivatives and excreted in human urine
AU Lodge, John K.; Ridlington, James; Leonard, Scott; Vaule, Heather; Traber,
Maret G.
CS Linus Pauling Institute, Oregon State University, Corvallis, OR,
97331-6512, USA
SO Lipids (2001), 36(1), 43-48
CODEN: LPDSAP; ISSN: 0024-4201
PB AOCS Press
DT Journal
LA English
CC 18-2 (Animal Nutrition)
AB Limited information is available regarding metab. of vitamin E forms, esp.
the tocotrienols. Carboxyethyl-hydroxychromans (.alpha.- and .gamma.-
CEHC) are human urinary metabolites of .alpha.- and
.gamma.-tocopherols, resp. To evaluate whether tocotrienols are also
metabolized and excreted as urinary **CEHC**, urine was monitored
following tocotrienol supplementation. Complete (24 h) urine collections
were obtained for 2 d prior to (baseline), the day of, and 2 d after human
subjects (n = 6) ingested tocotrienol supplements. The subjects consumed
125 mg .gamma.-tocotrienyl acetate the first week, then the next week 500
mg; then 125 mg .alpha.-tocotrienyl acetate was administered the third
week, followed by 500 mg the fourth week. Urinary .alpha.- and .gamma.-
CEHC were measured by high-performance liq. chromatog. with
electrochem. detection. Urinary .gamma.-**CEHC** levels rose about
four- to sixfold in response to the two doses of .gamma.-tocotrienol and
then returned to baseline the following day. Significant (P < 0.0001)
increases in urinary .alpha.-**CEHC** were obsd. only following
ingestion of 500 mg .alpha.-tocotrienyl acetate. Typically, 1-2% of
.alpha.-tocotrienyl acetates or 4-6% of .gamma.-tocotrienyl acetates were
recovered as their resp. urinary **CEHC** metabolites. A .gamma.-
CEHC excretion time course showed an increase in urinary .gamma.-
CEHC at 6 h and a peak at 9 h following ingestion of 125 mg

.gamma.-tocotrienyl acetate. In summary, tocotrienols, like tocopherols, are metabolized to CEHC; however, the quantities excreted in human urine are small in relation to dose size.

ST vitamin E metabolite tocotrienol urine
IT Urine

(.alpha.- and .gamma.-tocotrienols metab. to carboxyethyl-hydroxychroman derivs. and excretion in human urine)

IT 1406-18-4, Vitamin E 1721-51-3, .alpha.-Tocotrienol 14101-61-2, .gamma.-Tocotrienol

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(.alpha.- and .gamma.-tocotrienols metab. to carboxyethyl-hydroxychroman derivs. and excretion in human urine)

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L5 ANSWER 23 OF 41 CAPLUS COPYRIGHT 2003 ACS

AN 2000:895441 CAPLUS

DN 134:192659

TI Bioavailability and potency of natural-source and all-racemic .alpha.-tocopherol in the human: A dispute

AU Hoppe, P. P.; Krennrich, G.

CS Nutrition Research Station, BASF Aktiengesellschaft, Offenbach, 76877, Germany

SO European Journal of Nutrition (2000), 39(5), 183-193
CODEN: EJNUFZ; ISSN: 1436-6207

PB Steinkopff Verlag

DT Journal

LA English

CC 18-2 (Animal Nutrition)

AB Alpha-tocopherol occurs in nature as a single stereoisomer (RRR) while synthetic vitamin E is a mixt. of eight stereoisomers (all-racemic, all-rac). The presently accepted ratio of biopotency (RRR: all-rac) is 1.36, based on the fetal resorption test in rats. This ratio has been disputed for humans. Clin. endpoint studies in humans are lacking, but

plasma responses to RRR-and all-rac were measured in bioavailability studies. In nine studies comparing unlabeled forms, the ratio of plasma parameters (AUC, Cmax or steady-state concn.) concurred with the accepted ratio of biopotency within accepted bounds of equivalence. Four recent studies with simultaneous application of trideutero-RRR and hexadeutero-all-rac resulted in ratios of up to 2 for plasma, and of .apprx. 2.7 and .apprx. 3.4 for .alpha.-**CEHC** (a urinary metabolite) and umbilical cord plasma, resp. Because these results have been widely assumed to reflect the difference in biopotency, this has prompted a proposal to the Food and Nutrition Board, National Academy of Sciences, USA to change the biopotency factor to 2:1. We challenge the validity of bioavailability data in lieu of clin. endpoints. Because RRR and all-rac are not chem. identical and differ in plasma and tissue kinetics and metab., the ratio of bioavailability parameters does not reflect the ratio of biopotency. This needs to be detd. in adequately designed studies using clin. and biochem. endpoints. Until such studies have been performed it does not appear prudent to exchange the presently accepted ratio based on valid bioassays, albeit in a model animal, for another that is based on erroneous conclusions from human studies.

ST alpha tocopherol bioavailability human

IT Nutrition, animal

(bioavailability and potency of natural-source and all-racemic .alpha.-tocopherol in the human: A dispute)

IT 59-02-9, .alpha.-Tocopherol 10191-41-0, all-rac-.alpha.-Tocopherol
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(bioavailability and potency of natural-source and all-racemic .alpha.-tocopherol in the human: A dispute)

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L5 ANSWER 24 OF 41 CAPLUS COPYRIGHT 2003 ACS
AN 2000:788255 CAPLUS
DN 134:55952
TI Production of LLU-.alpha. following an oral administration of .gamma.-tocotrienol or .gamma.-tocopherol to rats
AU Hattori, Akihiro; Fukushima, Takeshi; Yoshimura, Hiroyuki; Abe, Kouichi; Imai, Kazuhiro
CS Department of Bio-Analytical Chemistry, Graduate School of Pharmaceutical Sciences, The University of Tokyo, Tokyo, 113-0033, Japan
SO Biological & Pharmaceutical Bulletin (2000), 23(11), 1395-1397
CODEN: BPBLEO; ISSN: 0918-6158
PB Pharmaceutical Society of Japan
DT Journal
LA English
CC 18-2 (Animal Nutrition)
Section cross-reference(s): 13
AB An oral administration of .gamma.-tocotrienol (.gamma.-T3) or .gamma.-tocopherol (.gamma.-Toc) to male rats caused an increase of the concn. of 2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxy chroman (LLU-.alpha., .gamma.-CEHC), a natriuretic compd., in plasma with a Tmax of 9 h. The configuration at C-2 of LLU-.alpha. produced from .gamma.-T3 or .gamma.-Toc was assigned as S-form by an HPLC equipped with a chiral column. These data indicated that LLU-.alpha. was produced not only from .gamma.-Toc but also .gamma.-T3, without racemization at C-2 in rats.
ST LLU tocotrienol tocopherol metab; chroman deriv formation tocotrienol tocopherol; hydroxychroman deriv formation tocotrienol tocopherol
IT 7616-22-0, .gamma.-Tocopherol 14101-61-2, .gamma.-Tocotrienol
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(LLU-.alpha. prodn. following an oral administration of .gamma.-tocotrienol or .gamma.-tocopherol to rats)
IT 178167-88-9
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(LLU-.alpha. prodn. following an oral administration of

.gamma.-tocotrienol or .gamma.-tocopherol to rats)
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structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
its structure diagram

FHITSEQ ----- First HIT RN, its text modification, its CA index name, its structure diagram, plus NTE and SEQ fields
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L5 ANSWER 25 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:759916 CAPLUS
 DN 134:36796
 TI .gamma.-Tocopherol and its major metabolite, in contrast to .alpha.-tocopherol, inhibit cyclooxygenase activity in macrophages and epithelial cells
 AU Jiang, Qing; Elson-Schwab, Ilan; Courtemanche, Chantal; Ames, Bruce N.
 CS Division of Biochemistry and Molecular Biology, University of California, Berkeley, CA, 94720, USA
 SO Proceedings of the National Academy of Sciences of the United States of America (2000), 97(21), 11494-11499
 CODEN: PNASA6; ISSN: 0027-8424
 PB National Academy of Sciences
 DT Journal
 LA English
 CC 1-7 (Pharmacology)
 AB Cyclooxygenase-2 (COX-2)-catalyzed synthesis of prostaglandin E2 (PGE2) plays a key role in inflammation and its assocd. diseases, such as cancer and vascular heart disease. Here we report that .gamma.-tocopherol (.gamma.T) reduced PGE2 synthesis in both lipopolysaccharide (LPS)-stimulated RAW264.7 macrophages and IL-1.beta.-treated A549 human epithelial cells with an apparent IC50 of 7.5 and 4 .mu.M, resp. The major metabolite of dietary .gamma.T, 2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxychroman (.gamma.-CEHC), also exhibited an inhibitory effect, with an IC50 of .apprxeq.30 .mu.M in these cells. In contrast, .alpha.-tocopherol at 50 .mu.M slightly reduced (25%) PGE2 formation in macrophages, but had no effect in epithelial cells. The inhibitory effects of .gamma.T and .gamma.-CEHC stemmed from their inhibition of COX-2 activity, rather than affecting protein expression or substrate availability, and appeared to be independent of antioxidant activity. .gamma.-CEHC also inhibited PGE2 synthesis when exposed for 1 h to COX-2-preinduced cells followed by the addn. of arachidonic acid (AA), whereas under similar conditions, .gamma.T required an 8- to 24-h incubation period to cause the inhibition. The inhibitory potency of .gamma.T and .gamma.-CEHC was diminished by an increase in AA concn., suggesting that they might compete with AA at the active site of COX-2. We also obsd. a moderate redn. of nitrite accumulation and suppression of inducible nitric oxide synthase expression by .gamma.T in lipopolysaccharide-treated macrophages. These findings indicate that .gamma.T and its major metabolite possess anti-inflammatory activity and that .gamma.T at physiol. concns. may be important in human disease prevention.
 ST gamma tocopherol cyclooxygenase 2 antiinflammatory
 IT Antioxidants
 (pharmaceutical; .gamma.-Tocopherol and its major metabolite inhibit

cyclooxygenase activity in macrophages and epithelial cells)

IT 39391-18-9
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (cyclooxygenase-2; .gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 59-02-9, .alpha.-Tocopherol
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 178167-88-9
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 7616-22-0, .gamma.-Tocopherol
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 363-24-6, PGE2 41598-07-6, PGD2 125978-95-2, Nitric oxide synthase
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 155976-51-5, 8-Isoprostane
 RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

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L5 ANSWER 26 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:728570 CAPLUS
 DN 134:16205
 TI Urinary .alpha.-tocopherol metabolites in .alpha.-tocopherol transfer protein-deficient patients
 AU Schuelke, Markus; Elsner, Angelika; Finckh, Barbara; Kohlschutter, Alfried; Hubner, Christoph; Brigelius-Flohe, Regina
 CS Department of Neuropediatrics, Charite University Hospital, Humboldt University Berlin, Berlin, D-13353, Germany
 SO Journal of Lipid Research (2000), 41(10), 1543-1551
 CODEN: JLPRAW; ISSN: 0022-2275
 PB Lipid Research, Inc.
 DT Journal
 LA English
 CC 14-14 (Mammalian Pathological Biochemistry)
 AB Patients with .alpha.-tocopherol transfer protein (.alpha.-TTP) defects experience neurol. symptoms characteristic of vitamin E deficiency and depend on continuous high .alpha.-tocopherol supplements. The authors investigated the excretion of 2,5,7,8-tetramethyl-2(2'-carboxyethyl)-6-hydroxychroman (.alpha.-CEHC), a urinary metabolite of .alpha.-tocopherol, as a putative marker for the .alpha.-tocopherol status of .alpha.-TTP-deficient patients and control subjects. In three patients vitamin E supplementation was stopped for short periods of time, during which plasma .alpha.-tocopherol concns. and urinary .alpha.-CEHC excretion were measured. In the patients, plasma .alpha.-tocopherol decreased below normal (<5 .mu.mol/l) but .alpha.-CEHC excretion remained above the range of unsupplemented control subjects (0.118-0.306

mg/day). In healthy subjects, however, .alpha.-CEHC excretion was increased only after surpassing a plasma .alpha.-tocopherol threshold of 30-40 .mu.mol/l. Such a threshold did not exist in patients. The general mechanism of .alpha.-tocopherol degrdn. did not appear to differ between patients and control subjects. The presumed mechanism of .omega.- and subsequent .beta.-oxidn. was supported by the detection of .alpha.-CPHC, an .alpha.-CEHC homolog with a side chain longer by 3 carbon atoms, both in supplemented patients and in control subjects.

ST

IT

Oxidation

(biol., .beta.- and .omega.-oxidn.; urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans in relation to)

IT

Biomarkers (biological responses)

Urine

(urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans)

IT

Transport proteins

RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(.alpha.-tocopherol transfer, deficiency; urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans)

IT

Blood plasma

(.alpha.-tocopherol; urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans)

IT

59-02-9, .alpha.-Tocopherol

RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans)

IT

4072-32-6

RL: ANT (Analyte); BOC (Biological occurrence); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence); USES (Uses)

(urinary .alpha.-tocopherol metabolite in .alpha.-tocopherol transfer protein-deficient (ataxia with isolated vitamin E deficiency) humans)

RE.CNT

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 AN 2000:456026 CAPLUS
 DN 133:149828
 TI Overview of studies on vitamin E metabolism - Missing link of vitamin E metabolism
 AU Nakamura, Tetsuya
 CS Department of Chemistry, Shibaura Institute of Technology, Fukasaku, Ohmiya, 330-8570, Japan
 SO Bitamin (2000), 74(5-6), 255-261
 CODEN: BTMNA7; ISSN: 0006-386X
 PB Nippon Bitamin Gakkai
 DT Journal; General Review
 LA Japanese
 CC 18-0 (Animal Nutrition)
 AB A review with 38 refs. Simon's metabolites (.alpha.-tocopheronic acid and its .gamma.-lactone) have long been considered the main metabolites of .alpha.-tocopherol in urine. A hypothetical pathway for the biol. formation of these metabolites was proposed via the tocopheryl quinone route. From the initial detection of a carboxyethyl hydroxychroman (CEHC)-type metabolite of .delta.-tocopherol (via the retention of the chroman ring route) in rat urine, corresponding metabolites of .alpha.-tocopherol and .gamma.-tocopherol were subsequently found. It is particularly interesting that .gamma.-CEHC (LLU-.alpha.) was found in human urine as a natriuretic factor. Whether Simon's metabolites are physiol. significant or are artifacts due to sample treatment is not certain at present. To identify them as conjugated forms is warranted. The lack of intermediary metabolites of vitamin E homologues still remains as a missing link in understanding the vitamin E catabolism.
 ST review vitamin E tocopherol intermediary metab
 IT Nutrition, animal
 (vitamin E and tocopherols intermediary metab.)
 IT Tocopherols
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (vitamin E and tocopherols intermediary metab.)
 IT 1406-18-4, Vitamin e
 RL: BPR (Biological process); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); PROC (Process); USES (Uses)
 (vitamin E and tocopherols intermediary metab.)

=> d 15 25 all

L5 ANSWER 25 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:759916 CAPLUS
 DN 134:36796
 TI .gamma.-Tocopherol and its major metabolite, in contrast to
 .alpha.-tocopherol, inhibit cyclooxygenase activity in macrophages and
 epithelial cells
 AU Jiang, Qing; Elson-Schwab, Ilan; Courtemanche, Chantal; Ames, Bruce N.
 CS Division of Biochemistry and Molecular Biology, University of California,
 Berkeley, CA, 94720, USA
 SO Proceedings of the National Academy of Sciences of the United States of
 America (2000), 97(21), 11494-11499
 CODEN: PNASA6; ISSN: 0027-8424
 PB National Academy of Sciences
 DT Journal
 LA English
 CC 1-7 (Pharmacology)
 AB Cyclooxygenase-2 (COX-2)-catalyzed synthesis of prostaglandin E2 (PGE2)
 plays a key role in inflammation and its assocd. diseases, such as cancer
 and vascular heart disease. Here we report that .gamma.-tocopherol
 (.gamma.T) reduced PGE2 synthesis in both lipopolysaccharide
 (LPS)-stimulated RAW264.7 macrophages and IL-1.beta.-treated A549 human
 epithelial cells with an apparent IC50 of 7.5 and 4 .mu.M, resp. The
 major metabolite of dietary .gamma.T, 2,7,8-trimethyl-2-(.beta.-
 carboxyethyl)-6-hydroxychroman (.gamma.-CEHC), also exhibited an
 inhibitory effect, with an IC50 of .apprx.30 .mu.M in these cells. In
 contrast, .alpha.-tocopherol at 50 .mu.M slightly reduced (25%) PGE2
 formation in macrophages, but had no effect in epithelial cells. The
 inhibitory effects of .gamma.T and .gamma.-CEHC stemmed from
 their inhibition of COX-2 activity, rather than affecting protein
 expression or substrate availability, and appeared to be independent of
 antioxidant activity. .gamma.-CEHC also inhibited PGE2
 synthesis when exposed for 1 h to COX-2-preinduced cells followed by the
 addn. of arachidonic acid (AA), whereas under similar conditions, .gamma.T
 required an 8- to 24-h incubation period to cause the inhibition. The
 inhibitory potency of .gamma.T and .gamma.-CEHC was diminished
 by an increase in AA concn., suggesting that they might compete with AA at
 the active site of COX-2. We also obsd. a moderate redn. of nitrite
 accumulation and suppression of inducible nitric oxide synthase expression
 by .gamma.T in lipopolysaccharide-treated macrophages. These findings
 indicate that .gamma.T and its major metabolite possess anti-inflammatory
 activity and that .gamma.T at physiol. concns. may be important in human
 disease prevention.
 ST gamma tocopherol cyclooxygenase 2 antiinflammatory
 IT Antioxidants
 (pharmaceutical; .gamma.-Tocopherol and its major metabolite inhibit
 cyclooxygenase activity in macrophages and epithelial cells)
 IT 39391-18-9
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (cyclooxygenase-2; .gamma.-Tocopherol and its major metabolite inhibit
 cyclooxygenase activity in macrophages and epithelial cells)
 IT 59-02-9, .alpha.-Tocopherol
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological
 study, unclassified); BIOL (Biological study)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase
 activity in macrophages and epithelial cells)
 IT 178167-88-9
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological
 study, unclassified); MFM (Metabolic formation); THU (Therapeutic use);
 BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase

activity in macrophages and epithelial cells)

IT 7616-22-0, .gamma.-Tocopherol
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 363-24-6, PGE2 41598-07-6, PGD2 125978-95-2, Nitric oxide synthase
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 155976-51-5, 8-Isoprostane
 RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
 (.gamma.-Tocopherol and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

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L5 ANSWER 35 OF 41 CAPLUS COPYRIGHT 2003 ACS
 AN 1999:437875 CAPLUS
 DN 131:198876
 TI Vitamin E: function and metabolism
 AU Brigelius-Flohe, Regina; Traber, Maret G.
 CS German Institute of Human Nutrition, Bergholz-Rehbrücke, D-14558, Germany
 SO FASEB Journal (1999), 13(10), 1145-1155
 CODEN: FAJOEC; ISSN: 0892-6638
 PB Federation of American Societies for Experimental Biology
 DT Journal; General Review
 LA English
 CC 18-0 (Animal Nutrition)
 AB A review with 112 refs. Although vitamin E has been known as an essential nutrient for reprod. since 1922, we are far from understanding the mechanisms of its physiol. functions. Vitamin E is a term for a group of tocopherols and tocotrienols, of which .alpha.-tocopherol has the highest biol. activity. Due to the potent antioxidant properties of tocopherols, the impact of .alpha.-tocopherol in the prevention of chronic diseases believed to be assocd. with oxidative stress has often been studied and beneficial effects have been demonstrated. Recent observations that the .alpha.-tocopherol transfer protein in the liver specifically sorts out RRR-.alpha.-tocopherol from all incoming tocopherols for incorporation into blood plasma lipoproteins, and that .alpha.-tocopherol has signaling functions in vascular smooth muscle cells that cannot be exerted by other forms of tocopherol with similar antioxidative properties, have raised interest in the roles of vitamin E beyond its antioxidative functions. The .gamma.-tocopherol may have functions apart from being an antioxidant. It is a nucleophile able to trap electrophilic mutagens in lipophilic compartments and it generates a metabolite that facilitates natriuresis. The metab. of vitamin E is equally unclear. Excess .alpha.-tocopherol is converted into 2,5,7,8-tetramethyl-2(2'-carboxyethyl)-6-hydroxychroman (.alpha.-CEHC) and excreted in urine. Other tocopherols, like .gamma.- and .delta.-tocopherol, are almost quant. degraded and excreted in the urine as the corresponding CEHCs. The all-rac-.alpha.-tocopherol compared to RRR-.alpha.-tocopherol is preferentially degraded to .alpha.-CEHC. Thus, there may be a specific mol. role of RRR-.alpha.-tocopherol that is regulated by a system that sorts, distributes, and degrades the different forms of vitamin E, but has not yet been identified. We try to summarize current knowledge on the function of vitamin E, with emphasis on its antioxidant vs. other properties, the preference of the organism for RRR-.alpha.-tocopherol, and its metab. to CEHCs.
 ST review nutrition vitamin E metab function

IT Nutrition, animal
(vitamin E nutritional function and metab.)
IT 1406-18-4, Vitamin e
RL: BPR (Biological process); BSU (Biological study, unclassified); FFD
(Food or feed use); BIOL (Biological study); PROC (Process); USES (Uses)
(vitamin E nutritional function and metab.)

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- (112) Wu, S; Biol Reprod 1973, V8, P625 CAPLUS

=> s cox 2

11379 COX

7755459 2
L6 4989 COX 2
(COX(W)2)

=> e skin

E1	3	SKIMSTOCK/BI
E2	1	SKIMSTOCKS/BI
E3	189684	--> SKIN/BI
E4	1	SKIN1/BI
E5	1	SKIN19/BI
E6	30	SKIN2/BI
E7	3	SKIN2TM/BI
E8	2	SKIN2ZK/BI
E9	1	SKIN2ZK1301/BI
E10	1	SKINA/BI
E11	4	SKINAKAS/BI
E12	6	SKINATH/BI

=> s e3

L7 189684 SKIN/BI

=> s l7 and l6

L8 170 L7 AND L6

=> d l8 140-170

L8 ANSWER 140 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:504149 CAPLUS
DN 131:252216
TI Chemopreventive activity of celecoxib, a specific cyclooxygenase-2 inhibitor, and indomethacin against ultraviolet light-induced **skin** carcinogenesis
AU Fischer, Susan M.; Lo, Herng-Hsang; Gordon, Gary B.; Seibert, Karen; Kelloff, Gary; Lubet, Ronald A.; Conti, Claudio J.
CS Science Park-Research Division, The University of Texas M. D. Anderson Cancer Center, Smithville, TX, 78957, USA
SO Molecular Carcinogenesis (1999), 25(4), 231-240
CODEN: MOCAE8; ISSN: 0899-1987
PB Wiley-Liss, Inc.
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 141 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:392167 CAPLUS
DN 131:39377
TI Cancer chemopreventive activity of resveratrol
AU Jang, M.; Pezzuto, J. M.
CS Dep. Surgical Oncology, Univ. Illinois, Chicago, IL, 60612, USA
SO Drugs under Experimental and Clinical Research (1999), 25(2/3), 65-77
CODEN: DECRDP; ISSN: 0378-6501
PB Bioscience Ediprint Inc.
DT Journal
LA English
RE.CNT 93 THERE ARE 93 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 142 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:330046 CAPLUS
DN 130:332886
TI Use of sesquiterpene lactones for treatment of severe inflammatory

disorders

IN Hwang, Daniel H.; Fischer, Nikolaus H.
PA Board of Supervisors of Louisiana State University and Agricultural and
Mechanical College, USA
SO U.S., 15 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	---	-----	-----	-----
PI	US 5905089	A	19990518	US 1998-59480	19980413
PRAI	US 1997-80224	A	19970414		

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 143 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:252443 CAPLUS
DN 131:97867
TI N-methyl D-aspartate induced mechanical allodynia is blocked by nitric
oxide synthase and cyclooxygenase-2 inhibitors
AU Dolan, Sharron; Nolan, Andrea M.
CS Division of Veterinary Pharmacology, Department of Veterinary Preclinical
Studies, University of Glasgow Veterinary School, Glasgow, G61 1QH, UK
SO NeuroReport (1999), 10(3), 449-452
CODEN: NERPEZ; ISSN: 0959-4965
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 144 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:249760 CAPLUS
DN 131:100522
TI Metabolic targets of cancer chemoprevention: Interruption of tumor
development by inhibitors of arachidonic acid metabolism
AU Marks, F.; Furstenberger, G.; Muller-Decker, K.
CS Tumor Cell Regulation, Department B 0500, German Cancer Research Center,
Heidelberg, D-69120, Germany
SO Recent Results in Cancer Research (1999), 151(Chemoprevention of Cancer),
45-67
CODEN: RRCRBU; ISSN: 0080-0015
PB Springer-Verlag
DT Journal; General Review
LA English
RE.CNT 109 THERE ARE 109 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 145 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:203288 CAPLUS
DN 131:39144
TI Pharmacodynamics and pharmacokinetics of tolafenamic acid in ruminating
calves: evaluation in models of acute inflammation
AU Lees, P.; McKellar, Q. A.; Foot, R.; Gettinby, G.
CS Department of Veterinary Basic Sciences, The Royal Veterinary College,
Hatfield, AL9 7TA, UK
SO Veterinary Journal (1998), 155(3), 275-288
CODEN: VTJRFP; ISSN: 1090-0233
PB Bailliere Tindall Ltd.
DT Journal
LA English

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 146 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:93913 CAPLUS
DN 130:306145
TI Resveratrol blocks eicosanoid production and chemically-induced cellular transformation: implications for cancer chemoprevention
AU Jang, Meishiang; Pezzuto, John M.
CS Program for Collaborative Research in the Pharmaceutical Science, and Department of Medicinal Chemistry and Pharmacognosy, College of Pharmacy, and Department of Surgical Oncology, College of Medicine, University of Illinois at Chicago, Chicago, IL, 60612, USA
SO Pharmaceutical Biology (Lisse, Netherlands) (1998), 36(Suppl.), 28-34
 CODEN: PHBIFC; ISSN: 1388-0209
PB Swets & Zeitlinger B.V.
DT Journal
LA English

RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 147 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:17363 CAPLUS
DN 130:232038
TI Effects of resveratrol on 12-O-tetradecanoylphorbol-13-acetate-induced oxidative events and gene expression in mouse **skin**
AU Jang, Meishiang; Pezzuto, John M.
CS College of Pharmacy, Department of Medicinal Chemistry and Pharmacognosy, Program for Collaborative Research in the Pharmaceutical Science, University of Illinois at Chicago, Chicago, IL, 60612, USA
SO Cancer Letters (Shannon, Ireland) (1998), 134(1), 81-89
 CODEN: CALEDQ; ISSN: 0304-3835
PB Elsevier Science Ireland Ltd.
DT Journal
LA English

RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 148 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1999:1230 CAPLUS
DN 130:204808
TI An analysis from clinicoepidemiological data of the principal adverse events from the COX-2-selective NSAID nimesulide, with particular reference to hepatic injury
AU Rainsford, K. D.
CS Biomedical Research Centre, Division of Biomedical Sciences, Sheffield Hallam University, Sheffield, S1 1WB, UK
SO Inflammopharmacology (1998), 6(3), 203-221
 CODEN: IAOAES; ISSN: 0925-4692
PB Kluwer Academic Publishers
DT Journal
LA English

RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 149 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:711828 CAPLUS
DN 130:79822
TI Pharmacological analysis of cyclooxygenase-1 in inflammation
AU Smith, Christopher J.; Zhang, Yan; Koboldt, Carol M.; Muhammad, Jerry; Zweifel, Ben S.; Shaffer, Alex; Talley, John J.; Masferrer, Jaime L.; Seibert, Karen; Isakson, Peter C.

CS Searle Research and Development, St. Louis, MO, 63198, USA
SO Proceedings of the National Academy of Sciences of the United States of America (1998), 95(22), 13313-13318
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 150 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:709449 CAPLUS
DN 129:310615
TI Possible background mechanisms of the effectiveness of cyclooxygenase-2 inhibitors in the treatment of rheumatoid arthritis
AU Katori, M.; Majima, M.; Harada, Y.
CS Department Pharmacology, School Medicine, Kitasato University, Japan
SO Inflammation Research (1998), 47(Suppl.2), S107-S111
CODEN: INREFB; ISSN: 1023-3830
PB Birkhaeuser Verlag
DT Journal
LA English

L8 ANSWER 151 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:700324 CAPLUS
DN 130:23533
TI Transcriptional regulation of cyclooxygenase-2 in mouse **skin** carcinoma cells: regulatory role of CCAAT/enhancer-binding proteins in the differential expression of cyclooxygenase-2 in normal and neoplastic tissues
AU Kim, Youngsoo; Fischer, Susan M.
CS Department of Carcinogenesis, M. D. Anderson Cancer Center, Science Park-Research Division, University of Texas, Smithville, TX, 78957, USA
SO Journal of Biological Chemistry (1998), 273(42), 27686-27694
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 152 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:638045 CAPLUS
DN 129:340137
TI Cholecalciferol induces prostaglandin E2 biosynthesis and transglutaminase activity in human keratinocytes
AU Kanekura, Takuro; Lalederkind, Stanley J. F.; Kirtikara, Kanyawim; Goorha, Sarita; Ballou, Leslie R.
CS Department of Medicine, College of Medicine, University of Tennessee, Memphis, TN, USA
SO Journal of Investigative Dermatology (1998), 111(4), 634-639
CODEN: JIDEAE; ISSN: 0022-202X
PB Blackwell Science, Inc.
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 153 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:575527 CAPLUS
DN 129:310592
TI Cyclooxygenase-2 inhibitor NS-398 improves survival and restores leukocyte

counts in burn infection

AU Shoup, Margo; He, Li-Ke; Liu, Hong; Shankar, Ravi; Gamelli, Richard
CS Loyola University Medical Center, Burn and Shock Trauma Institute and the
Department of Surgery, Maywood, IL, 60153, USA
SO Journal of Trauma: Injury, Infection, and Critical Care (1998), 45(2),
215-221
CODEN: JOTRFA; ISSN: 1079-6061
PB Williams & Wilkins
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 154 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:536774 CAPLUS
DN 129:240323
TI Activation of the epidermal platelet-activating factor receptor results in
cytokine and cyclooxygenase-2 biosynthesis
AU Pei, Yong; Barber, Lisa A.; Murphy, Robert C.; Johnson, Christopher A.;
Kelley, Susan W.; Dy, Lady C.; Fertel, Richard H.; Nguyen, Thanh M.;
Williams, David A.; Travers, Jeffrey B.
CS Departments Dermatology Pediatrics, Indiana University School Medicine,
Indianapolis, IN, 46202, USA
SO Journal of Immunology (1998), 161(4), 1954-1961
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
RE.CNT 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 155 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:502947 CAPLUS
DN 129:211426
TI Pharmacological evaluation of 1-(carboxymethyl)-3,5-diphenyl-2-
methylbenzene, a novel arylacetic acid with potential anti-inflammatory
properties
AU Cutler, S. J.; De Witt Blanton, C., Jr.; Akin, D. T.; Steinberg, F. B.;
Moore, A. B.; Lott, J. A.; Price, T. C.; May, S. W.; Pollock, S. H.
CS Dep. Pharmaceutical Sci., Mercer Univ., Atlanta, GA, 30341, USA
SO Inflammation Research (1998), 47(7), 316-324
CODEN: INREFB; ISSN: 1023-3830
PB Birkhaeuser Verlag
DT Journal
LA English

L8 ANSWER 156 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:483336 CAPLUS
DN 129:298092
TI Measurement of cyclooxygenase inhibition in vivo: a study of two
non-steroidal anti-inflammatory drugs in sheep
AU Cheng, Z.; Nolan, A. M.; Mckellar, Q. A.
CS Division of Veterinary Pharmacology, Department of Veterinary Preclinical
Studies, University of Glasgow, Glasgow, G61 1QH, UK
SO Inflammation (New York) (1998), 22(4), 353-366
CODEN: INFLD4; ISSN: 0360-3997
PB Plenum Publishing Corp.
DT Journal
LA English
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 157 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:345378 CAPLUS
DN 129:78548
TI **Cox-2** expression is induced by UVB exposure in human
skin: implications for the development of **skin** cancer
AU Buckman, ShaAvhree Y.; Gresham, Alane; Hale, Pamela; Hruza, George; Anast,
Jason; Masferrer, Jaime; Pentland, Alice P.
CS Washington University School of Medicine, St. Louis, MO, USA
SO Carcinogenesis (1998), 19(5), 723-729
CODEN: CRNGDP; ISSN: 0143-3334
PB Oxford University Press
DT Journal
LA English
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 158 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1998:236085 CAPLUS
DN 129:710
TI Budesonide epimer R or dexamethasone selectively inhibit
platelet-activating factor-induced or interleukin 1.beta.-induced DNA
binding activity of cis-acting transcription factors and cyclooxygenase-2
gene expression in human epidermal keratinocytes
AU Lukiw, Walter J.; Pelaez, Ricardo Palacios; Martinez, Jorge; Bazan,
Nicolas G.
CS Louisiana State University Medical Center, Neuroscience Center of
Excellence and Department of Ophthalmology, School of Medicine, New
Orleans, LA, 70112-2272, USA
SO Proceedings of the National Academy of Sciences of the United States of
America (1998), 95(7), 3914-3919
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 159 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1997:593773 CAPLUS
DN 127:260856
TI The co-culture of dermal fibroblasts with human epidermal keratinocytes
induces increased prostaglandin E2 production and cyclooxygenase 2
activity in fibroblasts
AU Sato, Takashi; Kirimura, Yoshiaki; Mori, Yo
CS Department of Biochemistry, School of Pharmacy, Tokyo University of
Pharmacy and Life Science, Tokyo, 192-03, Japan
SO Journal of Investigative Dermatology (1997), 109(3), 334-339
CODEN: JIDEAE; ISSN: 0022-202X
PB Blackwell
DT Journal
LA English

L8 ANSWER 160 OF 170 CAPLUS COPYRIGHT 2003 ACS
AN 1997:582514 CAPLUS
DN 127:242963
TI Effect of topically applied cyclooxygenase-2-selective inhibitors on
arachidonic acid- and tetradecanoylphorbol acetate-induced dermal
inflammation in the mouse
AU Puignero, Violant; Queralt, Josep
CS Unitat de Fisiologia, Facultat de Farmacia, Universitat de Barcelona,
Barcelona, 08028, Spain
SO Inflammation (New York) (1997), 21(4), 431-442

CODEN: INFLD4; ISSN: 0360-3997

PB Plenum
DT Journal
LA English

L8 ANSWER 161 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1997:514963 CAPLUS

DN 127:214791

TI Suppressive effects of tranilast on the expression of inducible
cyclooxygenase (COX2) in interleukin-1.β-stimulated fibroblasts

AU Inoue, Hajime; Ohshima, Hideo; Kono, Hiroyuki; Yamanaka, Miwa; Kubota,
Takako; Aihara, Masaki; Hiroi, Tomoko; Yago, Nagasumi; Ishida, Hirotomo

CS DEPARTMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, ST. MARIANNA UNIVERSITY
SCHOOL OF MEDICINE, KAWASAKI, 216, Japan

SO Biochemical Pharmacology (1997), 53(12), 1941-1944

CODEN: BCPA6; ISSN: 0006-2952

PB Elsevier

DT Journal

LA English

L8 ANSWER 162 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1997:334710 CAPLUS

DN 127:6195

TI Crosslinked silicones forming water- and oil-repellent soft films useful
in coatings and cosmetics

IN Iyanagi, Koichi

PA Pola Chemical Industries, Inc., Japan

SO Jpn. Kokai Tokkyo Koho, 14 pp.

CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 09071657	A2	19970318	JP 1996-19132	19960205
PRAI	JP 1995-183583		19950627		

L8 ANSWER 163 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1997:331870 CAPLUS

DN 127:6196

TI Crosslinked silicones forming water- and oil-repellent soft films useful
in coatings and cosmetics

IN Iyanagi, Koichi

PA Pola Chemical Industries, Inc., Japan

SO Jpn. Kokai Tokkyo Koho, 16 pp.

CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 09071656	A2	19970318	JP 1996-19131	19960205
PRAI	JP 1995-183580		19950627		

L8 ANSWER 164 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1997:114096 CAPLUS

DN 126:140839

TI Induction of cyclooxygenase-2 expression by peroxisome proliferators and
non-tetradecanoylphorbol 12,13-myristate-type tumor promoters in
immortalized mouse liver cells

AU Ledwith, Brian J.; Pauley, Cindy J.; Wagner, Linda K.; Rokos, Carrie L.;
Alberts, David W.; Manam, Sujata

CS Depts. of Genetic and Cellular Toxicology, Merck Res. Lab., West Point,
PA, 19486, USA

SO Journal of Biological Chemistry (1997), 272(6), 3707-3714
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

L8 ANSWER 165 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1997:48225 CAPLUS

DN 126:69716

TI Pharmacokinetics and pharmacodynamics of ketoprofen enantiomers in the
horse

AU Landoni, M. F.; Lees, P.

CS Facultad de Ciencias Veterinarias, Universidad Nacional de la Plata, La
Plata, 1900, Argent.

SO Journal of Veterinary Pharmacology and Therapeutics (1996), 19(6), 466-474
CODEN: JVPTD9; ISSN: 0140-7783

PB Blackwell

DT Journal

LA English

L8 ANSWER 166 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1996:657461 CAPLUS

DN 125:316599

TI A long-term study to evaluate the safety and efficacy of meloxicam therapy
in patients with rheumatoid arthritis

AU Huskisson, E. C.; Ghozlan, R.; Kurthen, R.; Degner, F. L.; Bluhmki, E.

CS St Bartholomews Hospital, London, EC1A 7BE, UK

SO British Journal of Rheumatology (1996), 35(Suppl. 1), 29-34
CODEN: BJRHDF; ISSN: 0263-7103

PB Oxford University Press

DT Journal

LA English

L8 ANSWER 167 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1996:305816 CAPLUS

DN 124:335404

TI Regulation of the arachidonic acid cascade by mouse **skin** tumor
promoters

AU Fischer, S. M.; Lo, H. -H.; Li, E.; Maldve, R. E.

CS MD Anderson Cancer Center, University Texas, Smithville, TX, USA

SO Proceedings of the International Cancer Congress, Free Papers and Posters,
16th, New Delhi, Oct. 30-Nov. 5, 1994 (1994), Volume 1, 25-29. Editor(s):
Rao, R. S. Publisher: Monduzzi Editore, Bologna, Italy.

CODEN: 62UYAO

DT Conference

LA English

L8 ANSWER 168 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1996:223285 CAPLUS

DN 124:285156

TI Cyclooxygenases in human and mouse **skin** and cultured human
keratinocytes: association of **COX-2** expression with
human keratinocyte differentiation

AU Leong, Jane; Hughes-Fulford, Millie; Rakhlin, Nina; Habib, Aida; Maclouf,
Jacques; Goldyne, Marc E.

CS Veterans Affairs Med. Cent., Univ. California, San Francisco, CA, 94121,
USA

SO Experimental Cell Research (1996), 224(1), 79-87
CODEN: ECREAL; ISSN: 0014-4827

PB Academic

DT Journal
LA English

L8 ANSWER 169 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1996:131356 CAPLUS

DN 124:228562

TI Role of nitric oxide and prostaglandins in lipopolysaccharide-induced increase in vascular permeability in mouse **skin**

AU Fujii, Emiko; Irie, Kaoru; Ogawa, Akira; Ohba, Ken-ichi; Muraki, Takamura
CS Department of Pharmacology, Tokyo Women's Medical College, 8-1 Kawada-cho, Shinjuku-ku, Tokyo, 162, Japan

SO European Journal of Pharmacology (1996), 297(3), 257-63

CODEN: EJPHAZ; ISSN: 0014-2999

PB Elsevier

DT Journal

LA English

L8 ANSWER 170 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1994:214522 CAPLUS

DN 120:214522

TI Inducible isoforms of cyclooxygenase and nitric-oxide synthase in inflammation

AU Vane, John R.; Mitchell, Jane A.; Appleton, Ian; Tomlinson, Annette; Bishop-Bailey, David; Croxtall, Jamie; Willoughby, Derek A.

CS William Harvey Res. Inst., St. Bartholomew's Hosp. Med. Coll., London, EC1M 6BQ, UK

SO Proceedings of the National Academy of Sciences of the United States of America (1994), 91(6), 2046-50

CODEN: PNASA6; ISSN: 0027-8424

DT Journal

LA English

=> d 18 140 all

L8 ANSWER 140 OF 170 CAPLUS COPYRIGHT 2003 ACS

AN 1999:504149 CAPLUS

DN 131:252216

TI Chemopreventive activity of celecoxib, a specific cyclooxygenase-2 inhibitor, and indomethacin against ultraviolet light-induced **skin** carcinogenesis

AU Fischer, Susan M.; Lo, Herng-Hsang; Gordon, Gary B.; Seibert, Karen; Kelloff, Gary; Lubet, Ronald A.; Conti, Claudio J.

CS Science Park-Research Division, The University of Texas M. D. Anderson Cancer Center, Smithville, TX, 78957, USA

SO Molecular Carcinogenesis (1999), 25(4), 231-240

CODEN: MOCAE8; ISSN: 0899-1987

PB Wiley-Liss, Inc.

DT Journal

LA English

CC 1-6 (Pharmacology)

Section cross-reference(s): 8

AB Epidemiol. and dietary studies suggest that nonsteroidal anti-inflammatory drugs (NSAIDs) reduce the risk of colon cancer, possibly through a mechanism involving inhibition of cyclooxygenase (COX)-2, which is overexpressed in premalignant adenomatous polyps and colon cancer. Because UV light (UV) can induce COX-2 and nonspecific NSAIDs can decrease UV-induced **skin** cancer, we evaluated the ability of two compds., celecoxib (a specific COX-2 inhibitor) and indomethacin (a nonspecific NSAID), to block UV-induced **skin** tumor development in SKH:HR-1-hrBr hairless mice. Mice fed 150 or 500 ppm celecoxib showed a dose-dependent redn.

(60% and 89%, resp.) in tumor yield. Indomethacin (4 ppm) reduced tumor yield by 78%. Although both acute and chronic UV exposure increased cell proliferation and edema, neither compd. reduced these parameters. In contrast, UV-induced prostaglandin synthesis in the epidermis was effectively blocked by both compds. UV-induced increases in COX-2 expression in skin were also not altered in any of the treatment groups. Similarly, tumors that constitutively express high levels of COX-2 displayed no redn. by treatment with celecoxib or indomethacin. The dramatic protective effects of celecoxib suggests that specific COX-2 inhibitors may offer a way to safely reduce the risk of skin cancer in humans.

- ST COX2 celecoxib NSAID indomethacin UV skin carcinogenesis
- IT Radioprotectants
 - Transformation, neoplastic
 - UV radiation
 - (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT Prostaglandins
 - RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 - (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis: epidermal prostaglandin synthesis inhibition)
- IT Skin
 - (epidermis; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis: epidermal prostaglandin synthesis inhibition)
- IT Skin, neoplasm
 - Skin, neoplasm
 - (inhibitors; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT Anti-inflammatory agents
 - (nonsteroidal; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT Antitumor agents
 - Antitumor agents
 - (skin; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT 39391-18-9
 - RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 - (2; COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT 53-86-1, Indomethacin 169590-42-5, Celecoxib
 - RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 - (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis)
- IT 363-24-6, Pge2
 - RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 - (COX-2 inhibitor celecoxib and NSAID indomethacin prevention of UV light-induced skin carcinogenesis: epidermal prostaglandin synthesis inhibition)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE
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```
=> s prostaglandin e2
      62067 PROSTAGLANDIN/BI
          1 SKIMSTOCKS/BI
L9      0 PROSTAGLANDIN SKIMSTOCKS/BI
          ((PROSTAGLANDIN(W)SKIMSTOCKS)/BI)
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```
=> s prostaglandin
L10     62067 PROSTAGLANDIN
```

```
=> s pge2
L11     23922 PGE2
```

```
=> s skin aging
      189684 SKIN
      147511 AGING
L12     894 SKIN AGING
          (SKIN(W)AGING)
```

=> s l11 and l12
L13 1 L11 AND L12

=> d l13

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AN 2002:408501 CAPLUS
DN 136:390772
TI Cosmetic method of treating skin
IN Blachford, Sarah Louise; Ginger, Rebecca Susan; Parmar, Preyesh; Rogers, Julia Sarah; Yates, Paula Rachel
PA Unilever P.L.C., UK; Unilever N.V.; Hindustan Lever Limited
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002041865	A1	20020530	WO 2001-EP13039	20011107
	W:				
				AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM	
	RW:			GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG	
	AU 2002020694	A5	20020603	AU 2002-20694	20011107
PRAI	GB 2000-28355	A	20001121		
	WO 2001-EP13039	W	20011107		
RE.CNT	4				
					THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 16:16:23 ON 25 JUN 2003)

FILE 'REGISTRY' ENTERED AT 16:16:34 ON 25 JUN 2003

L1 15 S CHEC
L2 5 S CEHC

FILE 'CAPLUS' ENTERED AT 16:18:12 ON 25 JUN 2003

L3 2 S L2
L4 2 S L2
L5 41 S CEHC
L6 4989 S COX 2
E SKIN
L7 189684 S E3
L8 170 S L7 AND L6
L9 0 S PROSTAGLANDIN E2
L10 62067 S PROSTAGLANDIN
L11 23922 S PGE2
L12 894 S SKIN AGING
L13 1 S L11 AND L12

=> s l10 and l12
L14 1 L10 AND L12

=> s 114 not 113
L15 1 L14 NOT L13

=> d 115

L15 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AN 2001:467920 CAPLUS
DN 135:60186
TI Activators of peroxisome proliferator activated receptors .alpha..beta. in
treatment of skin diseases with immunol. basis and graft rejection
IN Kippenberger, Stefan; Loitsch, Stefan M.; Bernd, August
PA Johann Wolfgang Goethe-Universitaet, Germany
SO Ger. Offen., 8 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10053003	A1	20010628	DE 2000-10053003	20001018
PRAI	DE 1999-19950286	A1	19991019		

=> e wrinkles

E1	2	WRINKLER/BI
E2	3	WRINKLERESISTANT/BI
E3	2006 -->	WRINKLES/BI
E4	1	WRINKLEY/BI
E5	1734	WRINKLING/BI
E6	6	WRINKLINGS/BI
E7	21	WRINKLY/BI
E8	1	WRINLING/BI
E9	1	WRINN/BI
E10	1	WRINT/BI
E11	1	WRIO/BI
E12	1	WRIPPING/BI

=> s e3-e5

2006 WRINKLES/BI
1 WRINKLEY/BI
1734 WRINKLING/BI
L16 3578 (WRINKLES/BI OR WRINKLEY/BI OR WRINKLING/BI)

=> d his

(FILE 'HOME' ENTERED AT 16:16:23 ON 25 JUN 2003)

FILE 'REGISTRY' ENTERED AT 16:16:34 ON 25 JUN 2003

L1 15 S CHEC
L2 5 S CEHC

FILE 'CAPLUS' ENTERED AT 16:18:12 ON 25 JUN 2003

L3 2 S L2
L4 2 S L2
L5 41 S CEHC
L6 4989 S COX 2
E SKIN
L7 189684 S E3
L8 170 S L7 AND L6
L9 0 S PROSTAGLANDIN E2
L10 62067 S PROSTAGLANDIN
L11 23922 S PGE2

L12 894 S SKIN AGING
L13 1 S L11 AND L12
L14 1 S L10 AND L12
L15 1 S L14 NOT L13
E WRINKLES
L16 3578 S E3-E5

=> s l8 and l16

L17 1 L8 AND L16

=> d l17

L17 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AN 2002:692067 CAPLUS
DN 138:61121
TI Effect of Glycolic Acid on UVB-Induced Skin Damage and
Inflammation in Guinea Pigs
AU Park, K. S.; Kim, H. J.; Kim, E. J.; Nam, K. T.; Oh, J. H.; Song, C. W.;
Jung, H. K.; Kim, D. J.; Yun, Y. W.; Kim, H. S.; Chung, S. Y.; Cho, D. H.;
Kim, B. Y.; Hong, J. T.
CS National Institute of Toxicological Research, Department of General
Toxicology, Korea Food and Drug Administration, Seoul, 361-763, S. Korea
SO Skin Pharmacology and Applied Skin Physiology (2002), 15(4), 236-245
CODEN: SPAPFF; ISSN: 1422-2868
PB S. Karger AG
DT Journal
LA English
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l10 and l15

L15 IS NOT VALID HERE

For an explanation, enter "HELP DISPLAY".

=> s l10 and l16

L18 6 L10 AND L16

=> d l18 1-6

L18 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 2002:692067 CAPLUS
DN 138:61121
TI Effect of Glycolic Acid on UVB-Induced Skin Damage and Inflammation in
Guinea Pigs
AU Park, K. S.; Kim, H. J.; Kim, E. J.; Nam, K. T.; Oh, J. H.; Song, C. W.;
Jung, H. K.; Kim, D. J.; Yun, Y. W.; Kim, H. S.; Chung, S. Y.; Cho, D. H.;
Kim, B. Y.; Hong, J. T.
CS National Institute of Toxicological Research, Department of General
Toxicology, Korea Food and Drug Administration, Seoul, 361-763, S. Korea
SO Skin Pharmacology and Applied Skin Physiology (2002), 15(4), 236-245
CODEN: SPAPFF; ISSN: 1422-2868
PB S. Karger AG
DT Journal
LA English
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 2000:592520 CAPLUS
DN 133:182713
TI Method and composition for promoting hair growth

IN Jones, Marcus R.
PA USA
SO PCT Int. Appl., 14 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000048559	A2	20000824	WO 2000-US3973	20000217
	WO 2000048559	A3	20001207		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 1999-252780	A1	19990219		

L18 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 1996:202114 CAPLUS
DN 124:311654
TI Effect of cool preservation on pulmonary arterial smooth muscle cells
AU Hall, Susan M.; Haworth, Sheila G.
CS Developmental Vascular Biology, Institute Child Health, London, WC1N 1EH, UK
SO American Journal of Physiology (1996), 270(3, Pt. 1), L435-L445
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English

L18 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 1992:484103 CAPLUS
DN 117:84103
TI Eicosanoid-mediated contractility of hepatic stellate cells
AU Kawada, Norifumi; Klein, Heike; Decker, Karl
CS Biochem. Inst., Univ. Freiburg, Freiburg/Br., D-7800, Germany
SO Biochemical Journal (1992), 285(2), 367-71
CODEN: BIJOAK; ISSN: 0306-3275
DT Journal
LA English

L18 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 1987:433809 CAPLUS
DN 107:33809
TI Dopamine attenuates the contractile response to angiotensin II in isolated rat glomeruli and cultured mesangial cells
AU Barnett, R.; Singhal, P. C.; Scharschmidt, L. A.; Schlondorff, D.
CS Albert Einstein Coll. Med., Bronx, NY, USA
SO Circulation Research (1986), 59(5), 529-33
CODEN: CIRUAL; ISSN: 0009-7330
DT Journal
LA English

L18 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 1986:419354 CAPLUS
DN 105:19354
TI Effects of leukotrienes on isolated rat glomeruli and cultured mesangial

cells
AU Barnett, Richard; Goldwasser, Philip; Scharschmidt, Linda A.; Schlondorff, Detlef
CS Dep. Med., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SO American Journal of Physiology (1986), 250(5, Pt. 2), F838-F844
CODEN: AJPHAP; ISSN: 0002-9513
DT Journal
LA English

=> s l6 and l16

L19 1 L6 AND L16

=> d l19

L19 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AN 2002:692067 CAPLUS
DN 138:61121
TI Effect of Glycolic Acid on UVB-Induced Skin Damage and Inflammation in Guinea Pigs
AU Park, K. S.; Kim, H. J.; Kim, E. J.; Nam, K. T.; Oh, J. H.; Song, C. W.; Jung, H. K.; Kim, D. J.; Yun, Y. W.; Kim, H. S.; Chung, S. Y.; Cho, D. H.; Kim, B. Y.; Hong, J. T.
CS National Institute of Toxicological Research, Department of General Toxicology, Korea Food and Drug Administration, Seoul, 361-763, S. Korea
SO Skin Pharmacology and Applied Skin Physiology (2002), 15(4), 236-245
CODEN: SPAPFF; ISSN: 1422-2868
PB S. Karger AG
DT Journal
LA English
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s cyclooxygenase

L20 17646 CYCLOOXYGENASE

=> s s l20 and l16

MISSING OPERATOR S L20

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s l20 and l16

L21 3 L20 AND L16

=> d l21 1-3

L21 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2003 ACS
AN 2002:692067 CAPLUS
DN 138:61121
TI Effect of Glycolic Acid on UVB-Induced Skin Damage and Inflammation in Guinea Pigs
AU Park, K. S.; Kim, H. J.; Kim, E. J.; Nam, K. T.; Oh, J. H.; Song, C. W.; Jung, H. K.; Kim, D. J.; Yun, Y. W.; Kim, H. S.; Chung, S. Y.; Cho, D. H.; Kim, B. Y.; Hong, J. T.
CS National Institute of Toxicological Research, Department of General Toxicology, Korea Food and Drug Administration, Seoul, 361-763, S. Korea
SO Skin Pharmacology and Applied Skin Physiology (2002), 15(4), 236-245
CODEN: SPAPFF; ISSN: 1422-2868
PB S. Karger AG
DT Journal
LA English

RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS
AN 2002:502829 CAPLUS
DN 137:68172
TI Pharmaceutical formulations of resveratrol for treatment of skin disorders
IN Pezzuto, John M.; Moon, Richard C.; Jang, Mei-Shiang; Ouali, Aomar; Lin, Shengzhao; Barillas, Karla Slowing
PA Pharmascience, Can.
SO U.S., 15 pp., Cont.-in-part of U.S. 6,008,260.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6414037	B1	20020702	US 1999-430337	19991029
	US 6008260	A	19991228	US 1998-5114	19980109
	WO 2001030336	A2	20010503	WO 2000-US41488	20001023
	WO 2001030336	A3	20011227		
	WO 2001030336	C2	20021227		
	W: CA, JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 1239849	A2	20020918	EP 2000-991709	20001023
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
	US 2002173472	A1	20021121	US 2002-71124	20020207
PRAI	US 1998-5114	A2	19980109		
	AU 1998-88420	A	19981009		
	US 1999-430337	A1	19991029		
	WO 2000-US41488	W	20001023		

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2003 ACS
AN 2001:870069 CAPLUS
DN 136:130852
TI Rays and arrays: the transcriptional program in the response of human epidermal keratinocytes to UVB illumination
AU Li, Deling; Turit, Thomas G.; Schuck, Alyssa; Freedberg, Irwin M.; Khitrov, Gregory; Blumenberg, Miroslav
CS The R. O. Perelman Department of Dermatology, New York University School of Medicine, New York, NY, USA
SO FASEB Journal (2001), 15(13), 2533-2535, 10.1096/fj.01-0172fje
CODEN: FAJOEC; ISSN: 0892-6638
PB Federation of American Societies for Experimental Biology
DT Journal
LA English

RE.CNT 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 121 2 3 all

L21 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS
AN 2002:502829 CAPLUS
DN 137:68172
TI Pharmaceutical formulations of resveratrol for treatment of skin disorders
IN Pezzuto, John M.; Moon, Richard C.; Jang, Mei-Shiang; Ouali, Aomar; Lin, Shengzhao; Barillas, Karla Slowing

PA Pharmascience, Can.
SO U.S., 15 pp., Cont.-in-part of U.S. 6,008,260.
CODEN: USXXAM

DT Patent
LA English
IC ICM A61K031-05

NCL 514733000

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 1

FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6414037	B1	20020702	US 1999-430337	19991029
	US 6008260	A	19991228	US 1998-5114	19980109
	WO 2001030336	A2	20010503	WO 2000-US41488	20001023
	WO 2001030336	A3	20011227		
	WO 2001030336	C2	20021227		
	W: CA, JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 1239849	A2	20020918	EP 2000-991709	20001023
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
	US 2002173472	A1	20021121	US 2002-71124	20020207
PRAI	US 1998-5114	A2	19980109		
	AU 1998-88420	A	19981009		
	US 1999-430337	A1	19991029		
	WO 2000-US41488	W	20001023		

AB A method is provided for preventing or treating skin conditions, disorders or diseases, such as may be assocd. with or caused by inflammation, sun damage or natural aging. The method involves administration, preferably topical administration, of an active agent selected from the group consisting of resveratrol, pharmacol. acceptable salts, esters, amides, prodrugs and analogs thereof, and combinations of any of the foregoing. Pharmaceutical formulations for use in conjunction with the aforementioned method, such as ointments, creams, lotions, and emulsions are provided as well. For example, a topical resveratrol compn. in the form of cream was prepd. contg. (by Wt.) polyethylene glycol and ethylene glycol palmitostearate 5%, caprilic/capric triglycerides 5%, oleoyl macrogol glycerides (Labrafil M 1944CS) 4%, cetyl alc. 5.5%, PPG-2 myristyl ether propionate (Crodamol PMP) 6%, xanthan gum 0.3%, water 48%, propylene glycol 1%, methylparaben 0.18%, propylparaben 0.02%, trans-resveratrol 10%, and diethylene glycol monoethyl ether (Transcutol) 15%. An off-white, stable cream was obtained. The cream inhibited wrinkle formation in hairless mice.

ST topical resveratrol antiinflammatory antitumor skin

IT Keratosis

(actinic; topical resveratrol formulations for treatment of skin disorders)

IT Skin, disease

(aging, wrinkles; topical resveratrol formulations for treatment of skin disorders)

IT Dermatitis

(atopic; topical resveratrol formulations for treatment of skin disorders)

IT Mammary gland, neoplasm

(chemoprevention of; topical resveratrol formulations for treatment of skin disorders)

IT Dermatitis

(contact; topical resveratrol formulations for treatment of skin disorders)

IT Lupus erythematosus

(discoid; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(emulsions; topical resveratrol formulations for treatment of skin disorders)

IT Skin, disease
(epidermolysis bullosa; topical resveratrol formulations for treatment of skin disorders)

IT Erythema
(erythema nodosum; topical resveratrol formulations for treatment of skin disorders)

IT Dermatitis
(exfoliative; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(gels; topical resveratrol formulations for treatment of skin disorders)

IT Radicals, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of formation of; topical resveratrol formulations for treatment of skin disorders)

IT Skin, neoplasm
(inhibitors; topical resveratrol formulations for treatment of skin disorders)

IT Skin
(keratinization; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(lotions; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(microemulsions; topical resveratrol formulations for treatment of skin disorders)

IT Erythema
(multiforme; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(ointments, creams; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(ointments; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(oral; resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(parenterals; resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(prodrugs; topical resveratrol formulations for treatment of skin disorders)

IT Antitumor agents
(promotion inhibitors; topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems
(solns.; topical resveratrol formulations for treatment of skin disorders)

IT Anti-inflammatory agents
Dermatomyositis
Mutation inhibitors
Psoriasis
Seborrhea
Skin, disease
Skin preparations (pharmaceutical)

Sunburn

(topical resveratrol formulations for treatment of skin disorders)

IT Drug delivery systems

(topical; topical resveratrol formulations for treatment of skin disorders)

IT 9032-20-6, Quinone reductase

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(induction of; topical resveratrol formulations for treatment of skin disorders)

IT 329900-75-6, **Cyclooxygenase 2** 329967-85-3,
Cyclooxygenase 1

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of; topical resveratrol formulations for treatment of skin disorders)

IT 501-36-0, Resveratrol 501-36-0D, trans-Resveratrol, analogs and derivs.
27208-80-6 61434-67-1, cis-Resveratrol 61434-67-1D, cis-Resveratrol,
conjugates with saccharides 148766-36-3

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(topical resveratrol formulations for treatment of skin disorders)

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

- (1) Anon; JP 61060609 1986 CAPLUS
- (2) Anon; JP 409328410 A 1997
- (3) Anon; JP 10045566 1998 CAPLUS
- (4) Anon; WO 9904747 1999 CAPLUS
- (5) Bertram; Cancer Lett 1979, V7, P289 CAPLUS
- (6) Cavazza; US 5747536 A 1998 CAPLUS
- (7) Cho; US 5411986 A 1995 CAPLUS
- (8) Gerhauser; Nature Med 1995, V1(3), P260 MEDLINE
- (9) Goodwin; Am J Med 1984, V77, P7 CAPLUS
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L21 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2003 ACS

AN 2001:870069 CAPLUS

DN 136:130852

TI Rays and arrays: the transcriptional program in the response of human epidermal keratinocytes to UVB illumination
 AU Li, Deling; Turit, Thomas G.; Schuck, Alyssa; Freedberg, Irwin M.; Khitrov, Gregory; Blumenberg, Miroslov
 CS The R. O. Perelman Department of Dermatology, New York University School of Medicine, New York, NY, USA
 SO FASEB Journal (2001), 15(13), 2533-2535, 10.1096/fj.01-0172fje
 CODEN: FAJOEC; ISSN: 0892-6638
 PB Federation of American Societies for Experimental Biology
 DT Journal
 LA English
 CC 8-7 (Radiation Biochemistry)
 AB The epidermis, our first line of defense from UV light, bears the majority of photodamage, which results in skin thinning, wrinkling, keratosis, and malignancy. Hypothesizing that skin has specific mechanisms to protect itself and the organism from UV damage, we used DNA arrays to follow UV-caused gene expression changes in epidermal keratinocytes. Of the 6,800 genes examd., UV regulates the expression of at least 198. Three waves of changes in gene expression can be distinguished, 0.5-2, 4-8, and 16-24 h after illumination. The first contains transcription factors, signal transducing, and cytoskeletal proteins that change cell phenotype from a normal, fast-growing cell to an activated, paused cell. The second contains secreted growth factors, cytokines, and chemokines; keratinocytes, having changed their own physiol., alert the surrounding tissues to the UV damage. The third wave contains components of the cornified envelope, as keratinocytes enhance the epidermal protective covering and, simultaneously, terminally differentiate and die, removing a carcinogenic threat. UV also induces the expression of mitochondrial proteins that provide addnl. energy, and the enzymes that synthesize raw materials for DNA repair. Using a novel skin organ culture model, we demonstrated that the UV-induced changes detected in keratinocyte cultures also occur in human epidermis in vivo.
 ST UVB radiation regulated gene skin keratinocyte
 IT Macrophage inflammatory protein 2
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (MIP-2.alpha.; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)
 IT Cell membrane
 Cytoskeleton
 DNA repair
 Post-transcriptional processing
 Signal transduction, biological
 (UVB-regulated genes in human epidermal keratinocytes)
 IT Chemokines
 Cytokines
 Elastins
 Gene, animal
 Growth factors, animal
 Interleukin 8
 Transcription factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (UVB-regulated genes in human epidermal keratinocytes)
 IT Transcription factors
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (c-myc; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)
 IT Cell envelope
 (cornified; UVB-regulated genes in human epidermal keratinocytes)
 IT Antioxidants
 (defense proteins; UVB-regulated genes in human epidermal keratinocytes)
 IT Cell junction

(desmosome; UVB-regulated genes in human epidermal keratinocytes)

IT Metabolism
(energy; UVB-regulated genes in human epidermal keratinocytes)

IT Skin
(epidermis; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(genes regulated by; UVB-regulated genes in human epidermal keratinocytes)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(involucrins; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Skin
(keratinocyte; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Chemokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(melanoma growth-stimulating activity-.beta.; transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Skin, disease
(photoaging; transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT Post-translational processing
(proteolytic; UVB-regulated genes in human epidermal keratinocytes)

IT DNA microarray technology
Human
UV B radiation
(transcriptional program in response of human epidermal keratinocytes to UVB illumination)

IT mRNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

IT Caseins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.beta.-; UVB-regulated genes in human epidermal keratinocytes)

IT 329900-75-6, **Cyclooxygenase 2**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(transcriptional program in response of human epidermal keratinocytes to UVB illumination: changes in protein and mRNA)

RE.CNT 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD
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(FILE 'HOME' ENTERED AT 16:16:23 ON 25 JUN 2003)

FILE 'REGISTRY' ENTERED AT 16:16:34 ON 25 JUN 2003

L1 15 S CHEC
L2 5 S CEHC

FILE 'CAPLUS' ENTERED AT 16:18:12 ON 25 JUN 2003

L3 2 S L2
L4 2 S L2

L5 41 S CEHC
 L6 4989 S COX 2
 E SKIN
 L7 189684 S E3
 L8 170 S L7 AND L6
 L9 0 S PROSTAGLANDIN E2
 L10 62067 S PROSTAGLANDIN
 L11 23922 S PGE2
 L12 894 S SKIN AGING
 L13 1 S L11 AND L12
 L14 1 S L10 AND L12
 L15 1 S L14 NOT L13
 E WRINKLES
 L16 3578 S E3-E5
 L17 1 S L8 AND L16
 L18 6 S L10 AND L16
 L19 1 S L6 AND L16
 L20 17646 S CYCLOOXYGENASE
 L21 3 S L20 AND L16

=> s l20 and l12

L22 2 L20 AND L12

=> d l22 1-2

L22 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
 AN 2001:800060 CAPLUS
 DN 136:66317
 TI UV erythema reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined in comparison to indomethacin
 AU Grundmann, Jens-Uwe; Bockelmann, Raik; Bonnekoh, Bernd; Gollnick, Harald P. M.
 CS Department of Dermatology and Venereology, Otto-von-Guericke-University, Magdeburg, D-39120, Germany
 SO Photochemistry and Photobiology (2001), 74(4), 587-592
 CODEN: PHCBAP; ISSN: 0031-8655
 PB American Society for Photobiology
 DT Journal
 LA English
 RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
 AN 2001:780665 CAPLUS
 DN 135:313633
 TI Treatment of hyperproliferative, inflammatory and related mucocutaneous disorders using inhibitors of mevalonate synthesis and metabolism
 IN Parks, Thomas P.; Grayson, Stephen
 PA Cellegy Pharmaceuticals, Inc., USA
 SO PCT Int. Appl., 50 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001078706	A2	20011025	WO 2001-US12175	20010412
	WO 2001078706	A3	20020328		
	WO 2001078706	C2	20021227		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
 CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
 HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,

LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
 SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
 YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
 BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 US 2002010128 A1 20020124 US 2001-833384 20010411
 PRAI US 2000-197357P P 20000413

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L22 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
 AN 2001:800060 CAPLUS
 DN 136:66317
 TI UV erythema reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined in comparison to indomethacin
 AU Grundmann, Jens-Uwe; Bockelmann, Raik; Bonnekoh, Bernd; Gollnick, Harald P. M.
 CS Department of Dermatology and Venereology, Otto-von-Guericke-University, Magdeburg, D-39120, Germany
 SO Photochemistry and Photobiology (2001), 74(4), 587-592
 CODEN: PHCBAP; ISSN: 0031-8655
 PB American Society for Photobiology
 DT Journal
 LA English
 CC 8-9 (Radiation Biochemistry)
 Section cross-reference(s): 1
 AB UV light exerts hazardous effects such as induction of skin cancer and premature **skin aging**. In this study we evaluated an assumptive anti-inflammatory effect of the nonsedative histamine H1-receptor antagonist, mizolastine, on UV-induced acute sunburn reaction. Therefore, a clin., randomized, double-blind, four-arm, crossover study was conducted in healthy young female volunteers (skin type II) comparing the UV sensitivity under mizolastine, acetyl-salicylic acid (ASA), indomethacin or a mizolastine/ASA combination. Moreover, HaCaT keratinocytes were incubated with mizolastine under various UV treatment modalities in vitro to study its effect on the release of inflammatory cytokines, i.e. interleukin (IL)-1.alpha., IL-6 and tumor necrosis factor .alpha. (TNF-.alpha.). All three drugs were effective in suppressing the UVB-, UVA- and combined UVA/UVB-erythema. However, the strongest effects were obsd. using the combined treatment with both 250 mg ASA and 10 mg mizolastine. An inhibitory effect in vitro of 10 nM mizolastine upon UV-induced cytokine release from HaCaT keratinocytes was obsd. for IL-1.alpha. at 24 h after 10 J/cm2 UVA1, for IL-6 at 48 h after 10 J/cm2 UVA1 and 30 mJ/cm2 UVB, and also for TNF-.alpha. at 4 h after 10 J/cm2 UVA, 10 J/cm2 UVA1 and 30 mJ/cm2 UVB, resp. The combination of mizolastine and ASA can be strongly recommended as a protective measure against UV erythema development with a lower unwanted side effect profile than that of the hitherto treatment modality, i.e. indomethacin.
 ST UV erythema protection antiinflammatory mizolastine acetylsalicylic acid cytokine
 IT Anti-inflammatory agents
 Drug interactions
 Erythema
 Human
 Radioprotectants
 Sunburn
 UV A radiation
 UV radiation
 (UV erythema-reducing capacity of mizolastine compared to acetyl-salicylic acid or both combined vs. indomethacin)

IT Interleukin 1.alpha.
Interleukin 6
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(UV erythema-reducing capacity of mizolastine compared to
acetyl-salicylic acid or both combined vs. indomethacin)

IT Skin, disease
(aging, prevention; UV erythema-reducing capacity of mizolastine
compared to acetyl-salicylic acid or both combined vs. indomethacin)

IT Cytokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inflammatory; UV erythema-reducing capacity of mizolastine compared to
acetyl-salicylic acid or both combined vs. indomethacin)

IT Skin
(keratinocyte; UV erythema-reducing capacity of mizolastine compared to
acetyl-salicylic acid or both combined vs. indomethacin)

IT 50-78-2, Acetyl-salicylic acid 108612-45-9, Mizolastine
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological
activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(UV erythema-reducing capacity of mizolastine compared to
acetyl-salicylic acid or both combined vs. indomethacin)

IT 39391-18-9, **Cyclooxygenase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; UV erythema-reducing capacity of mizolastine compared to
acetyl-salicylic acid or both combined vs. indomethacin)

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L22 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS

AN 2001:780665 CAPLUS

DN 135:313633

TI Treatment of hyperproliferative, inflammatory and related mucocutaneous disorders using inhibitors of mevalonate synthesis and metabolism

IN Parks, Thomas P.; Grayson, Stephen

PA Cellegy Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 50 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K031-00

ICS A61K031-366; A61K031-404; A61K031-22; A61K031-40; A61K031-575;
 A61K031-015; A61K031-196; A61K031-216; A61K045-06; A61P017-00;
 A61P029-00

CC 1-12 (Pharmacology)

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001078706	A2	20011025	WO 2001-US12175	20010412
	WO 2001078706	A3	20020328		
	WO 2001078706	C2	20021227		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
	US 2002010128	A1	20020124	US 2001-833384	20010411
PRAI	US 2000-197357P	P	20000413		

AB The invention provides methods for treating a variety of hyperproliferative and inflammatory mucocutaneous disorders, including basal cell carcinoma, squamous cell carcinoma, psoriasis and atopic dermatitis, as well as skin irritation and disorders assocd. with **skin aging** and skin photodamage using inhibitors of cholesterol metab. The invention further relates to the discovery that the combined use of several inhibitors of cholesterol metab. produces synergistic effects. Furthermore, the invention is directed to the use of inhibitors of cholesterol metab. as excipients to enhance the effects of antiinflammatory drugs.

ST mevalonate metab inhibitor antiproliferative antiinflammatory; cholesterol metab inhibitor antiproliferative antiinflammatory; mucocutaneous disorder mevalonate metab inhibitor; basal cell carcinoma mevalonate metab inhibitor; squamous cell carcinoma mevalonate metab inhibitor; psoriasis atopic dermatitis mevalonate metab inhibitor; skin irritation aging mevalonate metab inhibitor; photodamage skin mevalonate metab inhibitor

IT Cell proliferation
 (T cell; mevalonate synthesis and metab. inhibitors for treatment of

hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Skin, disease
(aging; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Peptides, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(analog; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Dermatitis
(atopic; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Dermatitis
(contact; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Skin, disease
(hyperproliferation; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Skin, disease
(irritation; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Anti-inflammatory agents
Dermatitis
Drug delivery systems
Drug interactions
Psoriasis
Skin, disease
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Corticosteroids, biological studies
Lanolin
Monoterpenes
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Drugs
Permeation enhancers
(mucocutaneous inflammation and irritation from; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Sterols
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oxysterols; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Skin, disease
(photoaging; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Skin, disease
(photodermatitis; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Proliferation inhibition
(proliferation inhibitors; mevalonate synthesis and metab. inhibitors

for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT T cell (lymphocyte)
(proliferation; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Prenylation
(protein prenylation inhibitor; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Drug interactions
(synergistic; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Drug delivery systems
(topical; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT Drug delivery systems
(transdermal, mucocutaneous inflammation and irritation from; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT 9028-35-7, HMG-CoA reductase 329900-75-6, **Cyclooxygenase 2**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT 150-97-0, Mevalonic acid
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT 57-88-5, Cholesterol, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT 53-86-1, Indomethacin 56-81-5, Glycerol, biological studies 64-86-8, Colchicine 69-72-7D, Salicylic acid, salicylates, biological studies 79-09-4, Propionic acid, biological studies 123-30-8, p-Aminophenol 536-59-4, Perillyl alcohol 566-27-8, 7.beta.-Hydroxycholesterol 2140-46-7, 25-Hydroxycholesterol 5989-27-5 7694-45-3, Perillic acid 22071-15-4, Ketoprofen 36322-90-4, Piroxicam 73573-88-3, Mevastatin 74103-06-3, Ketorolac 75330-75-5, Lovastatin 79902-63-9, Simvastatin 81093-37-0, Pravastatin 93957-54-1, Fluvastatin 132100-55-1, Dalvastatin 134523-00-5, Atorvastatin 145599-86-6, Cerivastatin 170006-72-1, FTI-276 171744-11-9, GGTI-286
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

IT 135371-29-8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(mevalonate synthesis and metab. inhibitors for treatment of hyperproliferative, inflammatory and related mucocutaneous disorders)

=> d his

(FILE 'HOME' ENTERED AT 16:16:23 ON 25 JUN 2003)

FILE 'REGISTRY' ENTERED AT 16:16:34 ON 25 JUN 2003

L1 15 S CHEC
L2 5 S CEHC

FILE 'CAPLUS' ENTERED AT 16:18:12 ON 25 JUN 2003

L3 2 S L2
L4 2 S L2
L5 41 S CEHC
L6 4989 S COX 2
E SKIN
L7 189684 S E3
L8 170 S L7 AND L6
L9 0 S PROSTAGLANDIN E2
L10 62067 S PROSTAGLANDIN
L11 23922 S PGE2
L12 894 S SKIN AGING
L13 1 S L11 AND L12
L14 1 S L10 AND L12
L15 1 S L14 NOT L13
E WRINKLES
L16 3578 S E3-E5
L17 1 S L8 AND L16
L18 6 S L10 AND L16
L19 1 S L6 AND L16
L20 17646 S CYCLOOXYGENASE
L21 3 S L20 AND L16
L22 2 S L20 AND L12

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
FULL ESTIMATED COST	ENTRY	SESSION
	153.37	168.26
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE	ENTRY	SESSION
	-8.46	-8.46

STN INTERNATIONAL LOGOFF AT 16:51:37 ON 25 JUN 2003